THE HEART

OLD AND NEW VIEWS

H. L. FLINT

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THE HEART:
OLD AND NEW VIEWS

THE HEART

OLD AND NEW VIEWS

BY

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WITH ILLUSTRATIONS

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TO

MY WIFE

AS A TOKEN OF APPRECIATION OF HER UNFAILING
ENCOURAGEMENT AND HELP

PREFACE

It was a happy notion of Dr. Flint to write a small book on the history of the heart from a clinical and anatomical point of view, and I shall be glad therefore if I can do the book any service—*quantum valeat*—by writing a few words of introduction. These can merely be of a commendatory kind, for it would be beyond the scope of the book to enter into its argument at any length. Both in the older history and in the new it seems to me that the author has succeeded in bringing together the salient points concerning both the doctrines of our ancestors and the researches of the most recent investigators. It is true, of course, that both the old matters and the new are to be found here and there in medical histories and modern textbooks; but, so far as I know, these have not been so selected and combined as to give a sharp picture of the whole. There is something very striking in thus setting forth in close neighbourhood the old and the new; in contrasting the tentative and speculative old doctrines with the volume of searching analysis of the most penetrating and systematic kind, which has distinguished the present generation. If there may be still a good deal yet to discover, yet we may feel encouraged by the progress made, and hope that we have attained at least to a plane of observation and reason high enough to enable us to treat diseases of the heart with insight and discrimination.

The heart, in so far as it is a machine—and such is its function to a large extent—has lent itself with peculiar success to mechanical methods of research; thus more precise knowledge has been won than in those fields of physiology where we meet with the more elusive problems of—let us say—colloid chemistry or internal secretion. To the elucidation of the working of the heart graphic methods are more applicable and more subject to instrumental precision. Of the more abstruse and intimate play of internal energy, such as the part of calcium in cardiac function, or the surface action of the muscular fibres apprehended by Professor Starling, there will be ere long much more to record; but for the present these phenomena are hardly in a phase advanced enough to lend themselves to exact demonstration.

As concerns "systolic murmurs," the subject of the last chapter, I think there is more to be said; some of it I have, however, published already, and I will not repeat it here. The central point, however, is whether or no the particular "systolic murmur" signifies mitral regurgitation? If it does, then energy is running to waste; and unless slight and stationary in degree must tell, sooner or later. The organ, however well and long it may run, did not start at scratch.

The reader will understand that Dr. Flint does not write this book merely as an historian or compiler; he writes not as one of the scribes; his own experience of heart problems during the war was considerable, so that what he says may be taken as the teaching of an expert. I cordially wish success to his little work.

CLIFFORD ALLBUTT.

CAMBRIDGE,
February, 1921.

INTRODUCTORY NOTE

In this study of the heart I have attempted to trace the steps which led from the darkness of the remote ages to the light of our present-day knowledge, and naturally, in so doing, I have dealt with the historical, physiological, and clinical aspects of the subject.

There is in our day an ever-increasing interest in the history of medicine, and I foresee that in the near future the mass of our knowledge will be so enlarged that it may necessitate an alteration in the methods of medical history, and that these which at present are of a general character will give place to the separate historical study of each individual organ.

It is true that the discovery of the circulation as a whole has been dealt with in great detail and the critical essays on the subject have been innumerable, but I do not find that there has been a consecutive history of the growth of heart knowledge from ancient to modern times.

This book is therefore brought forward to supply the need which I myself felt when my first interest in the subject was aroused, and I hope it may appeal to two classes of readers. In the first place it is intended for those who are interested in the heart but have neither access to the manifold literature on the subject nor time to sift the information it will yield. In the second place I hope it may be useful to those who wish to specialise in the subject, and for their benefit full side references are given.

It should be noticed that my method in this book has not been analytic but rather synthetic; I have attempted to piece together the fragments of information scattered throughout the centuries.

No knowledge is complete in itself: it is but a stage towards the far-off goal; thus the value of any new discovery can be appreciated only by comparing it with some past period of learning. So it is only by the study of the evolution of medicine that we can truly appraise the scientific knowledge of to-day, and realise its importance as a stepping-stone to paths of further discovery. So also it is only in studying the history of medicine that we come into contact with the life and work of the great pioneers of the past, and derive the enthusiasm and inspiration without which it is impossible to plough the long and lonely furrow of medical research.

It is not intended in this work to enter, in any detail, into either the prognosis or treatment of cardiac conditions; only the principles of treatment are touched upon.

I must gratefully acknowledge that I have drawn freely from the works of the authors referred to in both text and margin, and especially to the publications of Dr. Thomas Lewis and Sir James Mackenzie, from which I have taken my account and some diagrams of the more recent work on the mechanism of the heart-beat.

I wish to thank Dr. T. Wardrop Griffith for his kind permission to reproduce the electrocardiograms which I took while working under him at the Cardiological Centre of the Northern Command.

To the late Sir William Osler I am greatly indebted for valuable help and encouragement and for the trouble he took on my behalf.

I am under a great obligation to Sir Clifford Allbutt for his preface and for looking over my manuscript.

H. L. FLINT.

MANSFIELD,
March, 1921

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THE HEART
OLD AND NEW VIEWS

PART I

THE HEART IN ANTIQUITY

BEFORE describing in detail the process of the discovery of the anatomy and physiology of the heart, and more especially of the heart-beat, it may be interesting to refer to ancient times in order to find the position from which investigations started.

Reverting to very early Sumerian times (4000-3000 B.C.) we find that though the expression "heart" was largely used in a metaphorical sense, there was probably no clear distinction made between the heart and liver, the latter being the organ used for purposes of divination, presumably as the seat of the soul or divinity in the human body.

Hope, W. Hogg, "Heart and Reins," in the *Journal of the Manchester Oriental Society* 1911.

It was probably as a survival of Sumerian hepatoscopy that in Babylonian times the liver had the first place in primitive theology or physiology. This pre-eminence was obviously due to the bloody appearance of the liver, for man, from the earliest times, not only recognised blood as in a sense the essence of life, but he also assigned to it a divine power.

Professor Rhys Davids, writing on the Atharva Veda (1500 B.C.), states that though the literature of China, Chaldea, and Egypt, bears evidence of a belief in the soul residing in the heart, yet it contains no description

Rhys Davids in "Heart and Reins," *Ibid.*

of the form that soul was supposed to have; but in the Upanishads he found this interesting passage describing the soul:

"Theory of Soul in the Upanishads," by Rhys Davids, *J.R.A.S.*, 1899.

"In shape like a man, in size either as a grain of rice or a thumb, in appearance made (like the body) of the four elements (earth, water, air, and ether), of eyes, ears, and of certain mental qualities: this mannikin dwells in the heart." The Vedic priests held it to be the most sacred of mysteries, and earnestly considered means of preserving the soul in the heart, and of bringing it back if it should fly away during life. In order to ensure its safe-conduct to a better world when it flew away at death, they appear to have instituted the sacrifice, or ritualistic preservation, of the kidneys, by a process of reasoning which is somewhat obscure.

In this connection it is interesting to compare the very ancient Egyptian burial rites and customs. Our knowledge of these is partly derived from bas-reliefs and paintings which decorate the tombs of Egyptian nobles, and particularly from the scenes on the walls of the tombs in Western Thebes—*e.g.*, Seti I., Nineteenth Dynasty, and others belonging to the New Empire, 1600 to 600 B.C. During a short tour in April, 1916, I was much interested in seeing the tomb of Seti I., in which a whole series of funereal scenes is shown; in one of them is depicted the weighing of the heart of the deceased in a balance. A full explanation of this rite is to be found in the writings of Egyptologists such as Budge, Elliot Smith, Petrie, and others who have given most interesting descriptions and translations of the papyri Nebseni, Nu, Ani, Hunefer, and Anhai, which can be seen in the British Museum. The explanation is as follows: In the process of mummification most of the viscera were removed to the four canopic jars for preservation and placed beside the corpse. The heart was commonly supposed to have been included in those viscera, as in the case of the "Jersey Mummy" of the Twenty-first Dynasty; but Professor Elliot Smith has proved that the usual practice was to leave the heart in the body, and that any case of its removal was probably due to accident or to an error in the process of embalming. There is little doubt that the heart was treated separately from the liver, lungs, kidneys, etc., on account of the supreme importance attached to it; but for this very reason it was essential to find some means of guarding against its loss. In the "Book of the Dead" chapter xxvii. is entitled, "Whereby the heart of a person is not taken from him in the under-world." In order to secure the heart against such a catastrophe, a scarab was given to the deceased, placed on his breast, and supposed to accompany him on his journey. On the scarab was inscribed a part, or the whole, of a chapter from the "Book of the Dead" (generally chapter xxx.). A most interesting example is the scarab named the "Heart of Osiris," described by Petrie (No. 90 in "Amulets"); on it are written the following words: "My heart, of my Mother. My heart of my Mother, my heart of my becoming (in future life). May nothing rise up against me in evidence, may no hindrance be made against me by the divine chiefs, may there be no enemy of thee against me in the presence of the Guardian of the Balance. Thou art my Ka (likeness) in my body, the creator making sound my limbs . . ."; and he prays that "in the presence of the God before the great god of Amenti, there may be truth uttered"; and concludes: "Behold, thy uplifting is in the acquittal." This suggestion of a trial of the heart by weight is fully explained by reference to the papyrus Anhai, where we read: "At the other end of the Hall of Judgment is the

G. Elliot Smith in "Heart and Reins," in the *Journal of the Manchester Oriental Society*, 1911.

"Amulets," by W. M. F Petrie.

See also "Book of the Dead," by E. A. W. Budge

"Osiris and the Egyptian Resurrection," by E. A. W. Budge.

Balance, in which the heart of the deceased is to be weighed. . . . From a peg . . . hangs the beam of the Balance, with the two pans. The right pan usually holds the feather of Maat (=straight—*i.e.*, justice), or the goddess Maati herself, and on the left hand the heart which is to be weighed. The creature near the Balance is the monster Ammit, Eater of the Dead, with a head of a crocodile, the body of a lion, the hindquarters of a hippopotamus, who was believed to eat the hearts which were light (?) in the balance."

Professor Petrie, however, differs on this last point from Budge, for he believes that the just heart should not weigh heavier than the feather of righteousness; for he says in a letter which he wrote in answer to my question: "The heart is represented as weighed against the ostrich feather of the goddess of truth. As this was one of the lightest things probably evil would be shown by its being heavy. But I have never seen a drawing where the balance was tilted either way. The idea, therefore, was of its being exact." In any case it is noticeable that a man is to be judged before Osiris by the weight of his heart, and this can be taken as an example of the heart-soul theory.

In Hebraic literature we find traces of a similar line of thought, as in Ps. xxvi. (R.V.): "Try my reins and my heart"; Ps. vii. 9: "For the righteous God trieth the hearts and reins"; and Jer. xi. 20: "O Lord of hosts, that judgest righteously, that triest the reins and the heart." In all these passages the heart (in conjunction, that is, with the reins—*i.e.*, kidneys) is to be tested by Jahwe, to prove the state of the man's soul.

The Egyptian's great fear of the heart being stolen, and his belief in its indispensability in this life and in the hereafter, is frequently referred to in the "Book of the Dead." In chapter xxvii. the deceased apparently prays the Sons of Horus that the Stealers of Hearts may not take away his heart. In chapter xxix. B, "Let not my heart be taken from me, let it not be wounded, and may neither swords nor gashes be dealt upon me because it hath been taken from me;" and in chapter xxvi. we find this emphatic prayer: "May my heart be with me in the house of Hearts . . . and may it rest there, or I shall not eat of the cakes of Osiris."

"Book of the Dead," by E. A. W. Budge.

"Book of the Dead," chap. xxvi. (Pap. Ani).

A reminder of Egyptian funerary rites is found, by contrast, in the practice of "heart burial"—*i.e.*, the interring of the heart separately from the body. This custom was fairly common in medieval times in the case of royal personages—as, for instance, of Edward I. at Jerusalem, Robert the Bruce, and other persons. The hearts of the Hapsburgs were separately interred in the Loretto Chapel of the Augustiner Kirche at Vienna. In England the latest case on record is that of Paul Whitehead (in 1775), secretary to the "Monks" of the Medmenham Club.

"Chronicles of the Tombs," by Pettigrew.

From the ages of Egyptian antiquity one voice speaks with some true knowledge of heart function. In the papyrus discovered by Ebers at Thebes in 1872, a papyrus which dates back to about 1550 B.C. and contains incantations against disease and about seven hundred remedies, Nebsecht says: "The vessels (of the heart) lead to all the members . . . whether the doctor . . . lays his finger on the forehead, on the back of the head or on the hands, on the place of the stomach or on the feet, everywhere he meets with the heart (*i.e.*, the pulse ?) because its vessels lead to all the members. The heart is therefore called the beginning of all the members." Nebsecht's scientific knowledge must not, however, be overrated, and though

"Life in Ancient Egypt," by Erman, pp. 357 and 358.

these vessels correspond in their courses with the great arteries, he no doubt thought that they contained air, excretory fluids, etc. However, at all events he recognised that the vessels lead from the heart to the body.

THE HIPPOCRATIC PERIOD.

As primitive interest in anatomy developed and an elementary knowledge of the brain and heart was acquired, the heart-soul theory gradually gave way to a differentiation of bodily functions. To the brain was assigned intellectual power, to the heart the higher emotions such as love and courage, while to the liver were relegated the baser emotions of anger and jealousy. This seems to have been a theory generally established in Greece at the time of Plato. In his "Timæus" he makes the three rulers (ἀρχαί) of the body the head, heart, and liver, a view which, according to Galen, was held by Hippocrates. Before entering into any description of the anatomical knowledge of Hippocrates, the great Father of Medicine, it may be interesting to quote certain passages from the "Timæus" of Plato relating to the heart. These have little scientific value but great historical interest, as showing the workings of one of the greatest minds of the world in trying to understand the place of the human body in the universe. It must be remembered that Plato worked on an imaginary scheme rather than upon practical observation.

"Timæus" of Plato, by Archer Hind.

"De Plac. Hipp. et Plat.," vi. 505, by Galen.

Plato (428-347 B.C.).

He makes the heart the ruler of the arterial circulation, and the liver that of the venous; yet it is impossible to believe that he really distinguished between arteries and veins, as he nowhere else makes any such suggestion. His theory of the threefold soul is thus propounded:

"Timæus," by Archer Hind, xxxi., p. 258.

"And they (the children of God) in imitation of him, having received from him the immortal principle of soul, fashioned round about her a mortal body to ride in, and beside her they built in another kind of soul, even that which is mortal, having within itself dread and inevitable passions and pleasure . . . pain . . . confidence, wrath, and hope . . . reasonless sensation and love that ventures all things . . . and they set the mortal kind (the heart) to dwell apart from the other (the brain) in another chamber of the body, having built an isthmus or boundary between the head and the breast, setting the neck between them to keep them apart. So in the breast, or the thorax as it is called, they confined a mortal kind of soul. And whereas one part of it was nobler, the other baser, they built a wall across the hollow of the chest. . . . That part of the soul which shares courage and anger, seeing that it is so warlike, they planted nearer the head, between the midriff and the neck, that it might be within hearing of the reason and might join it in forcibly keeping down the tribe of lusts, when they would in no wise consent to obey the order and word of command from the citadel. And the heart which is the knot of veins and the fount of blood which rushes vehemently through all the limbs they made into a guardhouse, that whensoever the fury of anger boiled up at the message from the reason that some unrighteous dealing is being wrought around them . . . swiftly through all the narrow channels all the sensitive power in the body might be aware of the admonitions and threats, and be obedient to them and follow them altogether, and so permit the noblest part to be leader among them all. For the throbbing of the heart in the anticipation of danger . . . they devised . . . the lungs, which they set about the heart as it were a soft cushion to spring upon, *Ibid.*, xxxi., p. 255.

so that when wrath was at its height therein, the heart might leap upon a yielding substance and become cooled, and thus, being less distressed, it might, together with the emotions, be better enabled to obey the reason. But that part of the soul which lusts after meat and drink, and all things whereof it has need, owing to the body's nature, this they set between the midriff and the navel, and its boundary."

Ibid., xxxviii., p. 303.

We cannot enter here into a detailed description of Plato's views of the function of the heart, lungs, and blood, but it is interesting to notice that he regarded respiration as entirely subsidiary to digestion, a process accomplished by the action of fire in dividing the particles of food and imprinting its own hue upon them. "Whence the colour of the fluid that circulates through the body. . . . This we call Blood."

Alcmæon, 580 B.C.

Alcmæon of Croton, a contemporary of Pythagoras, believed, with others of the ancients, that sleep and waking are due to the ebb and flow of blood into the large vessels, the cessation of which causes death. He wrote several medical and philosophical works, to which Aristotle frequently refers, but they are lost. He is said to have dissected animals, and to have made important discoveries in anatomy and natural philosophy.

It must be remembered that Greek thought and medicine from the fifth century B.C.—and indeed through succeeding centuries—was dominated by the humoral theory. They regarded the world as consisting of the four elements earth, air, fire, and water—and their corresponding qualities, dry, cold, hot, and moist, and the four humours, blood, phlegm, black and yellow bile. This theory still holds good in Chinese medicine of the present day.

In the Hippocratic school we trace the foundation of clinical observation and the institution of scientific note-taking, which stand out in amazing contrast to the contemporary systems of other nations. The other schools, generally speaking, regarded physical and astronomic phenomena as divine, and therefore prohibited to the study of mortals.

Hippocrates, 460-370 B.C.

As Grote says, " Hippocrates merged into one the two classes of phenomena, the divine and the scientifically determinable, which Socrates had put asunder. Hippocrates treated all scientific phenomena as divine *and* scientifically determinable."

"History of Greece," by Grote.

It is probable that the Hippocratic school believed the heart to be the essence of life and the seat of the soul; and that the soul was immortal (unlike that later theory held by Plato of a "mortal soul in the thorax"). The heart, then, could not be diseased, for how could an immortal soul suffer injury ?

In stating the views of the school of Hippocrates upon the heart and its functions, and on the bloodvessels, we are faced with the difficulty of distinguishing between the genuine and the spurious Hippocratic writings. There is no doubt that the so-called Hippocratic treatise " De Corde" belongs to the latter class, and that neither the Father of Medicine nor his disciples composed it. It is a work which describes the heart with considerable accuracy as composed of a strong muscle, the pericardium as a smooth tunic containing a little fluid resembling urine, the auricles, which are compared to bellows, the ventricles, and the sigmoid valves. The writer speaks of the veins as originating in the heart, which is the fountain-head from which all parts of the body are irrigated, and he supposes that the left ventricle is the seat of the understanding.

"Genuine Works of Hippocrates," by Francis Adams. Pub. Sydenham Society, 1859.

Ibid., lib. i., 830.

"Reflections on Ancient and Modern Learning," by W. Wotton, 1697.

The treatise "De Morbis," probably composed by a disciple of Hippocrates, speaks of the "usual and constant motion of the blood," and this passage, amongst others, has been quoted by such writers as Vander-Linden, who tried to prove that Hippocrates, and not Harvey, was the true discoverer of the circulation. They also laid stress on the following passage:

"De Venis," 17.

"Reflections on Ancient and Modern Learning," chap. xviii.

"The bloodvessels, which are dispersed over the whole body, give spirit, moisture, and motion, and all spring from one, which (bloodvessel) has no beginning nor no end that I can find, for where there is a Circle there is no Beginning." But, as Wotton says, "It is plain from them that he did believe it as an hypothesis—that is, in plain English, that he did suppose Blood to be carried round the Body by a constant accustomed Motion. But that he did not know what the constant accustomed Motion was, and that he had not found that course which in our ages Dr. Harvey first clearly demonstrated, will appear evident."

In the Hippocratic (?) writing "De Structura Hominis" there is one passage which speaks of vessels as receiving blood from the heart and going to it, and another which says that blood is carried into the lungs from the heart for the nourishment of the lungs.

"Circulation," "Twentieth Century Practice of Medicine."

It is interesting to notice that palpation was not unknown to Hippocrates and his followers, for they would put a hand on the heart and feel its tumultuous beatings, mufflings and seat.

"De Historia Animalium," trans. by D'Arcy Wentworth-Thompson, book iii., 3, p. 512b. Oxf.: Clar. Press.

A striking treatise, "De Natura Humana," quoted by Galen and many other authors as genuine, is referred to by Aristotle as a work of Polybus, son-in-law to Hippocrates. Be that as it may, it contains a very interesting description of the four pairs of veins, all of which the author erroneously supposed to proceed from the head and neck. Aristotle quotes the passage and contradicts it, showing that bloodvessels originate in the heart, the shape and size of which he frequently and fairly accurately described.

Ibid., book iii., 3, 513a. and, book i., 17, 496a.

Aristotle 384–322 B.C

Aristotle spoke of the heart as a fount of blood, but supposed that the blood went from the heart to the veins, and gives a remarkable description of the heart as containing three cavities only: "It has three cavities, as has been said; the right-hand one is the largest of the three, the left-hand one the least, and the middle one intermediate in size. All these cavities, even the two smaller ones, are connected by passages within the lung, and this fact is rendered quite plain in one of the cavities. . . ." These cavities may correspond to the right ventricle, left ventricle, and left auricle respectively. This remarkable error, so called by many subsequent writers, including Galen and Cæsalpinus, is readily explained by Huxley's ingenious observation that Aristotle's numerous dissections were invariably performed on animals which had previously been killed by strangulation. Aristotle says: "The investigation of such a subject is fraught with difficulties, but if anyone be keenly interested in the matter, his best plan will be to allow his animals to starve to emaciation, then to strangle them on a sudden, and thereupon to prosecute his investigations." As a result of this process the great vein entering the right auricle would have been so distended as reasonably to give rise to the supposition of this auricle being merely a dilatation of the great vein. Hence Aristotle's account of the heart, which was so often repeated and discussed by later writers. It is also curious that he should have failed to observe the valves of the heart, but this easily explains his mistake in regarding the pulmonary artery as a branch of the great vein, since he was not aware of

Ibid., book i., 17, 496a. and book iii., 3, 513a, etc.

"Science and Culture," by T. H. Huxley, 1882.

"Historia Animalium," book iii., 3, 513a.

the tricuspid valves. For it was natural to think that the right auricle, when greatly distended, gave off the pulmonary artery. He described the bloodvessels as closely following the branches of the lung passages, and observed that they nowhere directly communicate one with another, but that air passed into the bloodvessels by a process of transudation or diffusion by means of the close proximity of the air passages and the bloodvessels. Unlike his successor, Erasistratus, he realised that all the heart cavities contained blood, and not air only, and his heart anatomy was surprisingly true for the times in which he lived. Huxley's defence of Aristotle in "Science and Culture" emphasises the importance of conceiving the conditions under which our predecessors laboured before we try to appraise the results of their efforts in research. If those critics who so frequently deplore the errors of Aristotle had themselves examined the heart of an animal killed by asphyxia, they might speedily have noted the appearance of the condition he so faithfully described.

Chap. viii.

Praxagoras.

Praxagoras of Cos is said to have been the first clearly to distinguish arteries from veins, stating that the former contain air only. Many believe, however, that this distinction was of much older date, and recognised even by the Ancient Egyptians. He was the first to use the pulse as an aid to diagnosis.

Herophilus (about 300 B.C.) and **Erasistratus.**

Herophilus of Chalcedon, a pupil of Praxagoras, and his contemporary Erasistratus, both of Alexandria, made important discoveries by the aid of dissection of man, a procedure which had been discountenanced by the priesthood. These two Alexandrine anatomists had the advantage of being under the benign government of the liberal-minded Ptolemies Soter, Philadelphus, and Euergetes. Herophilus was physician-in-ordinary to Ptolemy Soter, whose excessive zeal in anatomical experiments must have been very objectionable to a proportion of his less scientific subjects, if it is true that he used the bodies of unfortunate convicts as subjects for vivisection.

"Syriac Book of Medicine," by E. A. W. Budge, 1913

We know of the work of Herophilus and Erasistratus through Marx and Hieronymus and later writers, on whose authority it is shown that Erasistratus discovered the valves and chordæ tendinæ in the heart, and named them tricuspid and sigmoid. Though he rightly rejected the humoral theory accepted by Herophilus, he was equally erroneous in adopting the teaching of Praxagoras, who asserted that the arteries contained vapours only. In order to face the obvious objection that arteries bleed when wounded, he supposed that there must be communications between arteries and veins which, though usually closed, would allow blood to enter the arteries as soon as air escapes from them. Though he dissected he objected to the practice of venesection.

Herophilus was familiar with the pulse, and compared its variations to musical rhythms, and gave them specific names such as "goat-like" or bounding pulse—*i.e.*, "pulsus caprians" or "dicrotus," a name which still survives. He actually used a clepsydra, or water-clock, for timing the pulse, and he appears to have recognised the significance of pulse variations as a guide to the general condition of the body.

"Ein Beitragezur Geschichte der Medicin," vol. viii., pp. 104, by K. F. H. Marx. Carlsruhe u. Baden, 1838.

Celsus (about 31 B.C.–14 A.D.)

Celsus, the great medical historian of the time of Augustus Cæsar, showed an intimate acquaintance with the heart in his book "De Re Medicina."

THE GALENIC PERIOD.

Galen, A.D. 131–201.

“De Pulsibus ad Tyrones,” “De Differentus Pulsuum,” “De Diagnoscendis Pulsibus,” etc.

We must now pass forward to the Galenic period of medicine, which may be said to have lasted for 1,400 years. The writings of Galen of Pergamon have exerted greater influence for good or ill upon the succeeding practice of medicine than those of any other author. He was the most voluminous of the ancient writers, and amongst his works, which formed an encyclopædia of the knowledge of his time, are sixteen essays on the pulse, his pulse-lore being based upon the supposition of a specific pulse for each disease.

“De Venarum et Arteriarum Sectione,” by Galen; and see “De Fab. Human. Corp.,” lib. iii., cap. 5, by Vesalius, etc.

In anatomy he partly followed the Alexandrines, but for the most part his work was the outcome of his own dissection of monkeys and other animals. He described the muscles of the heart as longitudinal and transverse, the contraction of the former causing dilatation, and contraction of the latter narrowing, of the heart.

“De Usu Partium,” vi., xiii.

“De Usu Partium,” lib. vi., chap. viii., by Galen.

“Claudius Galenus,” by C. Victor Darenberg.

He observed that the heart when separated from all its vessels goes on beating, and he rightly concluded that the origin of motion lies within itself. In practice, then, Galen accepted the fact that the heart is muscular though he denied it in theory, as we see from the following: “The heart is made of hard flesh not easily injured, and is composed of various kinds of fibres, and although these two characteristics appear to give it the resemblance of muscles, it is evidently very different, for muscles are straight as regards their length and transverse in respect of their breadth. No muscle can have these two characteristics simultaneously in a reverse combination.”

“De Usu Partium,” vi., xiv.

“De Usu Partium,” vi., xxi., and “De Nat. Facult.” i. 7.

He insisted on diastole as being the principal motion of the heart, and likened this action to the bellows of a blacksmith. He ascribed this inherent power of attraction of the heart to its innate heat.

He described the valves of the heart, although a process of hypothetical reasoning led him to declare that they do not close perfectly. He also remarked upon the ductus arteriosus.

"De Usu Partium," vi., xi.

By the following experiments he overthrew the theory of Erasistratus that the arteries contain air only, and substituted his own doctrine of a mixture of blood and vital spirits. He says: "When an artery is wounded we always see blood escape, wherefore one of two things: Blood is either contained in the arteries immediately, or it flows into them from somewhere else. But if from elsewhere, it is obvious that the arteries in their natural state should contain spirit only. Were this the case, however, we should see spirit escape from a wounded artery as a prelude to any flow of blood; but as nothing of the kind occurs we conclude that the vessel never contained aught but blood"; and in an experiment: "If we lay bare an artery, include a portion of it between ligatures, and then open it, we shall find it full of blood."

"An Sanguis in Arteriis Natura Continеatur," by Galen; and "De Hipp. et Plato Dogm.," vii., ix.

Galen did not understand that the pulsation of the arteries is caused by the blood pumped into them by the heart, but taught that the arteries possessed a pulsative and attractive power inherent in their wall, arising from their bellows-like action of dilatation. He was convinced that the arteries originate in the heart, but the veins in the liver.

He knew of a communication between arteries and veins, for he says: "If you kill an animal by cutting many of its important arteries, you will find that the veins as well as the arteries are emptied of their blood, a thing which could not be unless the two orders of vessels inosculate." He did not, however, understand that blood as a whole

"De Hipp. et Plato Dogm.," vi., viii. 350.

is passed from an artery to a vein, but imagined that this anastomosis was merely a means whereby the arterial blood might receive nutriment from the venous, and return vital spirit in exchange: It was thus that he missed a cue to the discovery of the pulmonary circulation, although he recognised that some of the blood passed from the right side of the heart to the left by way of the lungs, for he says: " But all the blood thus intromitted is not for the nourishment of the heart; there is even a particular vein distributed to it for this purpose; and as there is no such disparity between the vena arteriosa and the arteria venosa, we cannot suppose that any considerable portion of the blood that is sent to the lungs, by the former vessel, is appropriated by them for their nourishment. It is therefore manifest that it must be transmitted to the left sinus of the heart." He, however, taught that the greater proportion of the blood in the right ventricle passed to the left, not through the lungs but by the pores in the septum. When writing on the attractive power of the diastole of the heart, he says " . . . It possesses in itself an inherent power of attraction; so that even as air is drawn into the lungs by the expansion of the chest in breathing, is the finer part of the blood attracted from the right to the left ventricle of the heart by its diastole, the partition between them having certain minute pores or orifices to this end designed. These, indeed, are seen with difficulty in the dead body, the parts being then cold, hard, and rigid. Reason assures us, however, that such pores must exist."

" De Usu Partium," lib. vi., chap. x.

Wm. Harvey, R. Willis.

" De Natura Facultatum," lib. i., cap. vii., Willis's translation.

He believed that every part of the body receives two distinct blood-supplies, one venous and the other arterial. In this connection he says: " The reason why there are two orders of vessels in the animal body is that the several parts may be supplied with the kind of nutriment appro-

" De Usu Partium," vi., xvii.

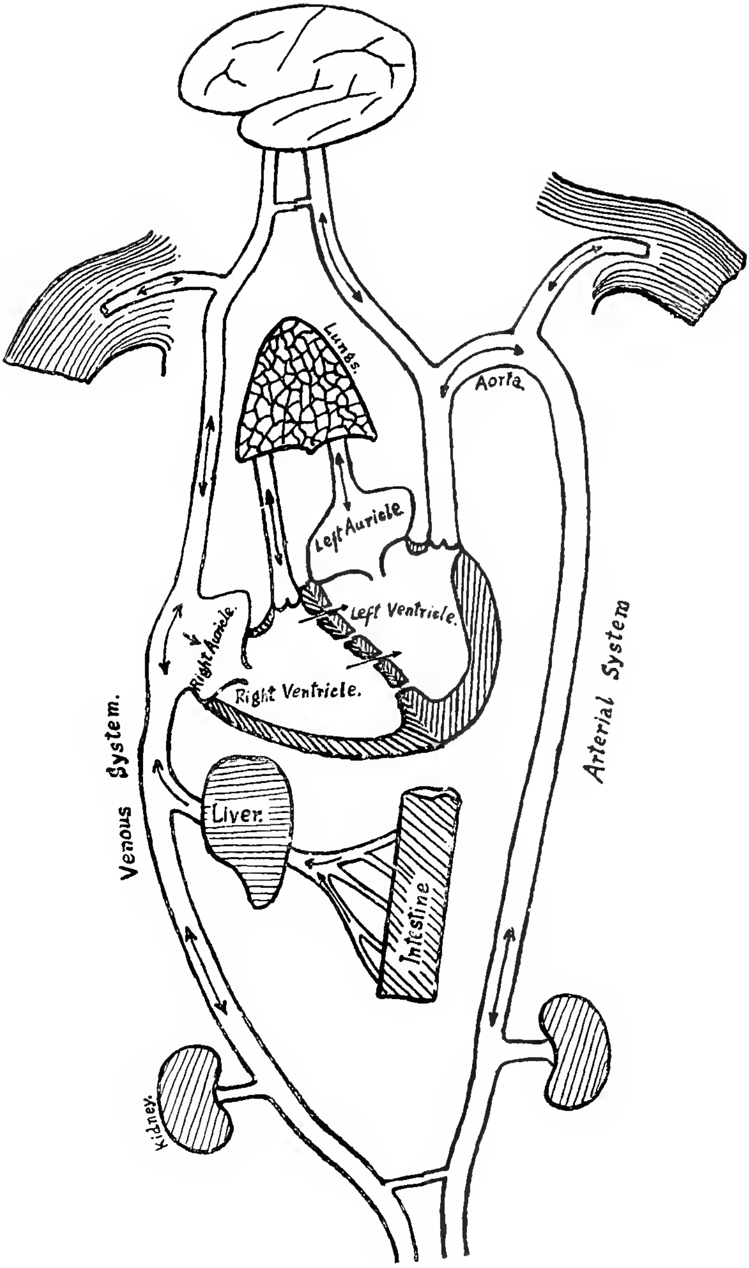

FIG. 1.—DIAGRAM ILLUSTRATING SOME OF GALEN'S VIEWS OF THE CIRCULATION.

(For explanation see opposite page.)

To face p. 17

priate to them. What so absurd as to suppose that the dense and heavy liver should have nourishment of the same kind imparted to it as the light and spongy lungs ?" He regarded the movement of the blood in both arteries and veins as a tidal ebb and flow. By an attractive force inherent in them, the organs of the body draw blood towards themselves, whilst the dilatation of the arteries and heart sucks the blood away from the organ and so produces the ebb of the tide.

We may now summarize Galen's conception of the circulation which I have attempted to represent diagrammatically in Fig. 1.

The food absorbed from the alimentary canal is carried by the vena porta to the liver, where it is converted into blood, and endowed with natural spirits. It then passes by way of the inferior vena cava into the great vein, part going into the right side of the heart. This blood, which is described as crude, and fit only to supply the lower functions of the organs, is carried to them by the veins, not in a continuous stream, but by a tidal ebb and flow. The crude blood in the right ventricle passes into the left, mainly through invisible pores in the septum, only a small portion going by way of the lungs. As the heart dilates, air or pneuma is drawn from the lungs

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Explanation of Diagram Illustrating Some of Galen's Views of the Circulation.

(*a*) The chyle is carried from the intestines to the liver and there converted into blood. (*b*) The veins originate in the liver. (*c*) There are two distinct blood systems—arterial and venous—the blood moves in each by a tidal ebb and flow. (*d*) Only a small part of the total venous blood enters the right auricle. (*e*) The greater part of that blood which enters the right side of the heart reaches the left ventricle by passing through small pores in the interventricular septum. (*f*) There are communications between the venous and arterial systems; this is not for the passage of blood from one into the other, but in order that the venous blood may give up nutriment to the arterial and in exchange receive vital spirit.

into the left ventricle by way of the pulmonary veins. In the left ventricle the crude blood, the air or pneuma, and the innate heat of the heart, combine to form blood endowed with vital spirits. This is regarded as fit to serve the higher functions of the organs which it reaches by way of the aorta and arteries, moving here also by an ebb and flow. This blood, on reaching the brain, is endowed with animal spirits, which is the highest form of spirit. During the endowment of crude blood with vital spirits, there are vapours produced which are given up into the lung passages during the ebb and flow of the blood in the pulmonary veins.

Although Galen in his writings laid the foundation of the physiology of the heart, his doctrine went far to hinder the discovery of the circulation, owing to his insistence on the four most misleading theories—

(1) That the origin of the veins is in the liver.

(2) That diastole is the most important movement of the heart.

(3) That the septum between the ventricles is permeable.

(4) That the motion of the blood in both arteries and veins is not in a continuous stream but by a tidal ebb and flow.

That the succeeding centuries were so devoid of discovery, not only of heart function, but also in other branches of medical science was due not so much to the theory of Galen as to his system, and to the general conditions of life and thought in the medieval period. For Galen founded not so much a science as a philosophic system, in which not the physician only but also the body of the patient were subject to decree, and regulated entirely by rote and dogma. Galen's numerous disciples lived and worked merely to perpetuate his doctrines, and did not use his methods and discoveries as stepping-stones to further knowledge. It must be admitted, however, that his influence and authority held the science of medicine intact through the dark and turbulent ages, when otherwise it might have fallen to pieces. Indeed, we should hardly expect to find rapid scientific progress in an age which in spiritual matters looked to an infallible Pope and the supreme domination of dogmatic creeds; but rather should we see how in the realm of medicine the medieval mind naturally leaned upon the authority of a dogmatist like Galen.

If I appear to have devoted too great a space to the writings of this one man, it is because they formed the basis of all discussions on cardiac function, even up to the seventeenth century and the time of Harvey.

From the time of Galen to the middle of the sixteenth century there was little original discovery in matters relating to the heart or circulation, but there were a few men whose references to the heart should not be overlooked.

I was interested in coming across "The Syriac Book of Medicine," by E. A. W. Budge, containing the text and a translation of the most important and most perfect Syrian treatise on anatomy, pathology, and therapeutics, which has come down to us. The manuscript, which is in his own possession, was written by a Syrian physician, probably a Nestorian, who translated it from the Greek. It is based upon the writings of Hippocrates, and is evidently post-Galenic, from the references to Galen's prescriptions. The following is an interesting reference to the clinical importance of the pulse: "From the heart, by means of the arteries, life is transmitted throughout the whole body, and that as it were by a kind of hands, the throbbing of the arteries which proceed from it maketh

Pub. 1913.

"The Syriac Book of Medicines," vol. ii., chap. xiv., p. 287, by E. A. W. Budge manifest to us all the diseases that happen in the body." The date of the Syrian manuscript is uncertain, but it must have been written during the early centuries of the Christian era, and it is a long stretch from this to the next author of any interest in connection with our subject. I refer to a French writer, Giles de Corbeil, Canon of Paris, and physician to Philip Augustus of France. He was of the Salernitan school, which in the twelfth century was still purely Hippocratic and Galenic in its teaching, and boasted of the title "City of Hippocrates," and had not then been invaded by Arabic medicine. Ægidius no doubt represents his school and his time, and his writings give an interesting picture of the practice of medicine and of the practitioner of his day. His writings are all in metrical form, not merely because that was the fashionable procedure, nor only with the object of augmenting his scholarly reputation, but chiefly for the very practical purpose of imprinting his teachings more firmly on the minds of his hearers.

Ægidius Corboliensis, 1165-1213.
"Giles de Corbeil," lib. ii., chap. ii., and iii., by Vieillard. Paris. 1909.

It must be remembered that the medical student frequently could not read, or, even if he could read, writing was impossible to him owing to his poverty and the high price of parchment. So the verses of the master were imparted orally to his pupils in the lecture-room, and formed the only handbook to anatomy and medicine which the student possessed. Ægidius wrote an amusing poem, "De Adventu Medici ad Ægrotum," containing much practical advice as to bedside manner and the other necessary qualifications for successful practice (or quackery, according to one's standpoint); but this is really an older book which he put into verse. The writing which concerns us now, however, is the tract "De Pulsibus," in which we find a table that shows an almost modern method of investigating the pulse. The follow-

See MS. Digby in the Bodleian Library, Oxford.

ing table taken from MS. Digby, Bodleian Library, Oxford, gives a translation of the headings under which he wrote his short pithy verses.

CONCERNING THE PULSE AND ITS SIGNS.

On a Large Pulse.
In what does a Large Pulse consist ?
Definition of a Small Pulse.
Strong or Weak Pulse.
Slow or Fast Pulse.
Hard or Soft Pulse.
Empty or Full Pulse.
Hot or Cold Pulse.
Frequent or Rare Pulse.
Pulsus Decidens.
Pulsus Incidens.
Equal Pulse.
Unequal Pulse.
Regular Pulse
Irregular Pulse.

He discusses the significance of these various conditions in great detail.

Mundinus

Mondino de Luzzi, otherwise Mundinus, is sometimes regarded as the founder of modern anatomy, but he was apparently overlooked by his immediate successors. His book, "Anatomia," completed in 1316, but published first in 1478 (at Padua), has on the title-page an interesting representation of the dissection of the human body. Though the book perpetuated many Galenic errors, it is important as having been the only textbook on anatomy for over 100 years. Mondino's description of the heart is surprisingly accurate in some respects, and he gives so remarkable a description of the valves that one can only feel astonished that he and his immediate successors did not go a few steps farther and grasp their true significance in relation to the pulmonary circulation. It should be clearly understood that Mundinus (like Aristotle) regarded the heart as being composed of three cavities only, corresponding to the right ventricle, left ventricle, and left auricle, respectively. The following is a literal translation of Mondino's description of the tricuspid and pulmonary valves: ". . . Then will appear the right ventricle, and you will there see two mouths, one of which is in the direction of the liver, and is the opening through which proceeds the chyle vessel (vena chilis) and it is a very big orifice, for through it the heart draws blood from the liver and sends blood to all the other members. Now, since through this opening more blood must be drawn in than is expelled, nature has ordained that it shall be shut at the time of contraction when the blood is expelled, and that it shall be open when the heart is dilated; and it has three valves which open from the outside to the inside. Now, through that orifice there is also an expulsion of perfectly concocted blood, all of which may not be expelled, for only one portion of it is sent through the lung, the other portion being converted into a spirit. Nature has therefore ordained that the doors be not lowered too far, and that they shall not be entirely closed. . . . Then indeed there is in the direction of the lung another opening, that of the arterial vein (pulmonary artery), which carries blood to the lung from the heart. For whereas the lung, on the one hand, attends to the needs of the heart by recompensing it, so, on the other hand, the heart sends blood to the lung, by this vein which is called the arterial vein. It is a vein, because it carries blood, and it has the nature of an artery because it hath two tunics. . . . In that opening, then (the pulmonary artery), you can see that there are three valves which open from the inside to the outside, and close perfectly from the outside to the

"Anothomia Mundini," by Joannes de Ketham. Ed. 1495.

inside, and which should not allow any blood to return at the time of dilatation (diastole)."

Later he describes the valves on the left side of the heart—*i.e.*, the mitral and aortic valves—with equal distinctness. An excellent commentary on the work of Mondino was written by Berenger, or Giacomo Berengario da Carpi, of Bologna, published in 1514 and 1521. Following Mondino's example, he dissected over 100 corpses, and his teaching was largely the outcome of personal observation; but his influence was not great, for he was driven from Bologna by the Church on the grounds that he had desecrated the "sacred human corpse." He appears to have been the first to cast doubts upon the permeability of the septum.

Leonardo da Vinci, 1452–1519

Leonardo da Vinci was a man far in advance of his time, and his work is gradually assuming its due place in medical history. He exerted little influence upon his time, for he did not publish his works, and his manuscripts, in mirror writing, were only discovered accidentally many years after his death. He studied on two lines, one being to read the published works of the authorities, the other that of personal observation, which he represented by remarkable drawings. Especially fine are those of the heart, on the strength of which he is now being acclaimed as the father of anatomy in the place of Vesalius. Leonardo recognised the advantage of a good drawing over any verbal description, for he says: "O writer, how can you adequately describe in words the entire configuration, as is done in the drawing? Your description is confused, lacking in knowledge, and you can convey but little knowledge of the true forms to the listener; and you are mistaken when you think that you can satisfy him by talking on any solid object which is surrounded by planes. Remember not to get involved by verbal

"Leonardo da Vinci," p. 5, by Arnold C. Klebs, 1916.

explanations unless you address the blind. . . . With what words will you describe the heart, without writing a book and without confusing the mind of the hearer by the very minuteness and thoroughness of your description?" We are naturally inquisitive as to his views on the permeability of the interventricular septum. He speaks of the septum as a colander or strainer, but in none of his drawings does he represent it as permeable. Klebs, however, believes, on the evidence of Leonardo's drawings, that he doubted the permeability of the septum, and that the references in his writings to its permeability were probably jottings from his book-reading, meant for further reference and verification. He refers to the coronary vessels as "vessels which nourish the heart," and gives a very full and accurate description of the valves. He depicts in his drawings the moderator band, and assigns to it the function of preventing over-distension. In a diagram he shows the relation of the ramifications of the bronchi to the bloodvessels, and he says: "To me it seems impossible that any air can penetrate into the heart through the bronchi." There is no doubt that he was much in advance of his time in his views on heart function. It is evident from his description in the "Quaderni d'Anatomia" that he realised that the function of the contraction of the heart is to drive blood into the arteries. In speaking of some observations he had made on the movements of a pig's heart, he says: "The heart shortens itself during its expulsion of the blood." Leonardo's work has become better known since the Scandinavian publication of a translation of his "Quaderni d'Anatomia."

Quad. ii., fol. 8, verso.

Sylvius, 1478–1555.

Jacques or Jacobus Sylvius, a bigoted Galenist, the teacher of Vesalius in Paris, exercised considerable influence on his age, since he was very popular as a lecturer, as many as 500 students being usually present. He was the first to describe the foramen ovale in the fœtal heart, and is credited with knowing of the existence of valves in the veins.

John Winter, 1487–1574.

John Winter of Andernach, another of Vesalius's teachers, calls for passing comment. We meet him first at Leyden as Greek master to the youthful Vesalius, and later at Paris as his teacher in anatomy. He describes the muscles of the heart as longitudinal, oblique, and transverse, and is in agreement with Sylvius in his description of their function—*i.e.*, the contraction of the longitudinal fibres by approximating the apex and base causes diastole, that of the transverse fibres gives rise to systole, whilst those which lie obliquely suspend their action momentarily, and secure the discharge of the fuliginous vapours from the blood into the lungs. He gives a good description of the valves of the heart, and explains that the reason why there are only two cusps in the mitral valve, whilst there are three in the tricuspid, is because the valves on the left side of the heart are not required to close so accurately as those on the right; so that the fuliginous vapours may escape and thus prevent the heart from becoming overheated. Amongst his numerous writings are many translations of the works of Galen. It is interesting to note that Servetus acted with Vesalius as prosector to Winter in Paris.

THE VESALIAN PERIOD.

With the sixteenth century and the work of Vesalius and his contemporaries we arrive at a new epoch in the history of medicine; yet it is noticeable that the influence of the Renaissance, and the invention of printing in the fifteenth century, did not become predominant in the world of medieval science until the seventeenth century and the time of Harvey. The Vesalian period, however, is of great importance, for it marks the birth of new methods and the revival of practical observation and original research, and the decay of philosophic rhetoric and the blind speculation which had so long survived on the base of Galenic authority.

Vesalius, 1515–1564.

The father of modern anatomy is Vesalius, a Fleming by birth, but of German extraction, who studied in Paris under Sylvius, that most bigoted of Galenists. The young student soon displayed an original personality by his vehement objection to the unscientific methods of dissection then in vogue. Paris at this time was at least a hundred years behind the Italian school and the example of Mondino, whose work had apparently not been noticed even by Etienne; and the dissection of the human body for purposes of demonstration, being repulsive to the fastidious master, was imperfectly performed by his clumsy barber servants. On one occasion, indeed, Vesalius was so exasperated by the inefficiency of such a demonstration that, starting from his seat and pushing aside the barbers, he seized their instruments and carried out the dissection with his own hands. His life work, it may be said, was done at Padua, where he held the chair of anatomy at the early age of twenty-two, and where his great book "De Fabrica Humani Corporis" was published in 1543. All the work on structure therein contained was based on his own personal dissection, which he performed with untiring zeal, even under the most trying conditions amounting at times to danger; for in Paris especially he was face to face with the great opposition of the Church to any tampering with the human body. It is true that in Italy under a republic and the protection of the Senate he had a freer hand than could have been the case in France, Belgium, or Spain. The difference between his principles and those of the Galenists may be commented upon in the words of Hippocrates: "Science and belief (opinion) are two things; the one begets knowledge, the other ignorance," for the attitude of Vesalius towards medical science is clearly expressed in his own words, when he says that he "studied that true bible as we count it (of) the human body, and the nature of man."

"History of Physiology," by Michael Foster.

Before describing his discoveries in matters connected with the heart and circulation, it is enlightening to examine his attitude towards Galenic doctrine. His own explanation found in his book is as follows: "I will expose without shrinking Galen's wrong statements about structure, but when I have to quote his views on function I will expound them without attacking them. I will content myself with letting drop here and there a hint of distrust and doubt." This may perhaps explain the fact that his anatomical descriptions of the heart were so exceedingly accurate and the illustrations in his book so wonderful, and yet that in questions of function he did not openly reject the Galenic theory of the permeability of the septum. It may well be surmised that Vesalius arrived nearer the truth than appears in his published work, if we remember that so many of his writings and illustrations perished at his own hands, as a consequence of the opposition, derision, and even persecution, to which his book "De Fabrica" had subjected him. In any case it is evident from the following ironical passage that he criticised the Galenic doctrine of heart function as early as 1543, in his first edition. He writes: "The septum of the ventricles, then, . . . made, as I have said, of the thickest substance of the heart, is on both sides plentifully endowed with little pits im-

"De Fab. Hum. Corp.," lib. vi., chap. xi. Eds. 1543 and 1546.

pressed upon it. Of these pits none, that is so far as can be discovered by the senses, can penetrate from the right to the left ventricle; whence we are forced to marvel at the work of the Creator of all things; by which the blood is made to sweat from the right to the left ventricle by pathways or passages which are invisible." As we shall presently see, he expresses his doubts more clearly in a later edition. He carefully explains that he has brought his statements into line with the Galenic doctrine, because in so great a work he is chary of enforcing new opinions, and does not "dare to swerve a nail's breadth from the doctrines of the Prince of Medicine." *Ibid.*, Præfatio.

Turning to his descriptions, we find that Vesalius determined the position of the heart in the chest, studied its construction, and ascertained that there are fibrous rings at the junction of auricles and ventricles. He described the valves and the columnæ carneæ and the musculi papillares; and his views on the structure of the ventricular walls were in agreement with those of his masters, Sylvius and Winter. *Ibid.*, lib.vi., chap. ix. *Ibid.*, vi.

He retained the Galenic theory of two independent circulations, right and left or venous and arterial, in both of which blood ebbed and flowed from and to the organs it supplied. *Ibid.*, vii., chap. xix.

Though Vesalius attracted enthusiastic pupils in flocks to his lectures at Padua, his book had so poor a reception that in bitter disappointment he threw up his chair and all prospects of further research and accepted the post of court physician to Charles V., with whom he remained until the last year or two before his early death at the age of fifty. During that time he republished his book "De Fabrica," and it is noticeable that in the edition of 1555 he more clearly defines his doubts as to the permeability of the septum than in the earlier edition of 1543; for his later words are: ". . . Moreover, it seemed to me most obscure by what motions the septum of the ventricles could be permeated. Yet by their interpretation during the process of dissection in the past, they were entirely convinced that blood is taken into the left ventricle from the right. How this could be performed was to my mind more than doubtful . . . and I will elsewhere speak more openly on the subject." It is conceivable that he might in the interval have read in manuscript (1546) Servetus' book, "De Restitutione Christianismi," which entirely rejects the hypothesis that blood passes from one ventricle to the other through the septum. In this book—which was not published, it must be noticed, till 1553—Servetus clearly describes the transition of blood by "an unknown route."

"De Fab. Hum. Corp.," Ed. Basil, lib. vi., cap. xi., p. 734.

Michael Servetus, 1509–1553.

Miguel Serveto, or Michael Servetus, fled from the Spanish Inquisition in his youth and went to Paris, where he studied under Sylvius and Günther. He was primarily a theologian, and his interest in human structure lay in his desire to know not only the human body, but from that the human spirit, and finally the nature of God as revealed in man. His books "De Trinitatibus" (1551), and more especially "De Restitutione Christianismi" (1553), diverged so far from orthodox theology that they aroused the burning indignation of the Church on the one hand and of Calvin on the other, and it was at the instigation of Calvin that he was burned at the stake in Geneva in 1553. There perished with him a thousand copies of the "Restitutio" which had been printed privately at Vienna; only three copies are known to have survived. His influence on the medicine of his own time must have been small, judging by the fact that the earliest commentary on his now famous description of the pulmonary

“Reflections on Ancient and Modern Learning,” chap. xviii., by W. Wotton, 1694.

“De Restitutio Christianismi,” lib. v., “De Trinitate,” p. 170, ed. 1553, and “Michael Servetus,” by William Osler.

circulation was not made until 140 years later—that is, by William Wotton in 1694, whose attention was drawn to it by Charles Bernard, a St. Bartholomew's Hospital surgeon. In the following passage he not only describes the pulmonary circulation, but also observes that the change in the character of the blood on the left side of the heart from that on the right is effected during its passage through the lungs, and not in the left ventricle. After mentioning the threefold spirit of the body of man—natural, vital, and animal—he goes on to discuss the vital spirit, and in a few paragraphs describes the pulmonary circulation: “Rightly to understand the question here, the first thing to be considered is the substantial generation of the vital spirit—a compound of the inspired air with the most subtle portion of the blood. The vital spirit has, therefore, its source in the left ventricle of the heart, the lungs aiding most essentially in its production. It is a fine attenuated spirit, elaborated by the power of heat, of a crimson colour and fiery potency—the lucid vapour, as it were, of the blood, substantially composed of water, air, and fire; for it is generated, as said, by the mingling of the inspired air with the more subtle portion of the blood which the right ventricle of the heart communicates to the left. This communication, however, does not take place through the septum, partition, or mid-wall of the heart, as commonly believed, but by another admirable contrivance, the blood being transmitted from the pulmonary artery (vena arteriosa) to the pulmonary vein (arteria venosa) by a long passage through the lungs, in the course of which it is elaborated, and becomes of a crimson colour. Mingled with the inspired air in this passage, and freed from fuliginous vapours by the act of expiration, the mixture being now complete in every respect, and the blood become fit dwelling-place of the vital spirit, it is finally attracted by the diastole, and reaches the left ventricle of the heart.

"Now that the communication and the elaboration take place in the lungs in the manner described, we are assured by the conjunctions and communications of the pulmonary artery with the pulmonary veins. The great size of the pulmonary artery seems of itself to declare how the matter stands; for this vessel would neither have been of such size as it is, nor would such a force of the purest blood have been sent through it to the lungs for their nutriment only; neither would the heart have supplied the lungs in such fashion seeing as we do that the lungs in the fœtus are nourished from another source—those membranes or valves of the heart not coming into play until the hour of birth, as Galen teaches. The blood must consequently be poured in such large measure at the moment of birth from the heart to the lungs for another purpose than the nourishment of these organs. Moreover, it is not simply air, but air mingled with blood that is returned from the lungs to the heart by the pulmonary veins.

"It is in the lungs, consequently, that the mixture (of the inspired air with the blood) takes place, and it is in the lungs also, not in the heart, that the crimson colour of the blood is acquired. There is not, indeed, capacity of room enough in the left ventricle of the heart for so great and important an elaboration, neither does it seem competent to produce the crimson colour. To conclude, the septum or middle portion of the heart, seeing that it is without vessels and special properties, is not fitted to permit and accomplish the communication and elaboration in question; although it may be that some transudation takes place through it It is a mechanism similar to that by which the transfusion from the vena porta to the vena cava takes place in the liver, in respect of the blood, that the transfusion from the pulmonary artery to the pulmonary vein takes place in the lungs, in respect of the spirit."

Realdus Columbus, 1516–1577.

The credit of Serveto's discovery is often given to Columbus, immediate successor to Vesalius in Padua. In 1559 "De Re Anatomica" was published, in which Columbus distinctly announced the circular course of the blood through the lungs as a discovery of his own in the following words: ". . . But they greatly erred from the true way, for blood is carried by the pulmonary artery to the lungs and is there refined and then, together with air, is carried to the pulmonary vein to the left ventricle of the heart, *a fact which no one before this time hath discovered or left to us in writing.*"

"De Re Anatomica," lib. vii.; "De Corde et Arteriis," p. 179. Ed. Venetiis, MDLIX.

Ibid., lib. vii.

He had no doubt as to the impossibility of the permeable septum, and he distinctly traced the course of the blood from the right to the left side of the heart through the lung. He denied that the heart was muscular, and he had no idea of a systemic circulation, but retained the old notion of an ebb and flow. In spite of his own assertion to the contrary, it is probable that his information about the pulmonary circulation was cribbed from Servetus, whose book "De Restitutione" was in manuscript as early as 1546, and it is known that one copy was undoubtedly sent to Curio at Padua.

Ibid., lib. xi., cap. ii.

Arantius, 1530–1589.

Before passing on to Cæsalpinus we should mention Arantius as the discoverer of the little bodies like milletseeds which exist in the semilunar valves. He pointed out that the perfect closure of the valves is due to these "corpora Arantii."

Constantius Varolius, 1543–1575.

We may also notice Varolius, physician to Pope Gregory XIII., and professor at Bologna. Like Servetus, he anticipated Harvey in appealing to the circulation in the fœtus in order to understand the passage of the blood from the right to the left side of the heart. In his "Anatomiæ," first published in 1559, he says: "In what way the blood passes from this (*i.e.*, the right) to that (*i.e.*, the left) ventricle not anyone has been able to demonstrate. In order to understand it, it is necessary to have a knowledge of the distribution of the blood in the fœtus, which I shall describe in another discourse."

"Anatomiæ," lib. ii., cap. i., p. 54. Ed. Francofurte, MDXCIX.

Andreas Cæsalpinus, 1519-1603.

A very interesting figure of the period is Cæsalpinus, that argumentative writer who is claimed as the discoverer of the circulation by those members of the Italian school who do not award that distinction to their other compatriot, Carlo Ruini, founder of modern veterinary medicine.

Cæsalpinus in his two publications, "Quæstiones Peripateticæ" (1571) and "Quæstiones Medicæ" (1593), certainly showed himself in advance of most of his contemporaries.

The following is a striking passage from his "Quæstiones Medicæ": "The passages of the heart are so arranged by nature that from the vena cava a flow takes place into the right ventricle, whence the way is open into the lungs. From the lungs, moreover, there is another entrance into the left ventricle of the heart, from which then a way is open into the aorta artery, certain membranes being so placed at the mouths of the vessels that they prevent return. There is a kind of perpetual movement through the heart and lungs into the aorta artery, as I have explained in my 'Quæstiones Peripateticæ.'"

"Quæstiones Medicæ," 2, xvii., p. 234. Second ed., MDXCIII.

"Quæstiones Peripateticæ," lib. v., cap. iv., p. 125. Ed. MDXCIII.

This Italian school holds that he knew something of the systemic circulation because he called attention to the fact that when a vein is ligatured it swells on the far side, whilst if blood be passed from the heart to the body by the veins the veins would swell on the near side when ligatured.

"Quæstiones Medicæ," 2, xvii., p. 234.

In reviewing the work of the old anatomists and physiologists, we must guard against reading into some isolated statement our present-day knowledge of the subject; and any claim to original discovery made for them by their successors must be judged in the light of their work as a whole, and not merely by some isolated sentence.

“Quæstiones Peripateticæ,” lib. v., cap. iv., p. 125. Ed. MDXCIII.

“Quæstiones Medicæ,” lib. ii., cap. xvii.

Ibid.

It is quite certain that Cæsalpinus in using the word “circulation” (“. . . huic sanguinis circulationi”) did not understand the systemic circulation as described by Harvey. He is Galenic in believing that veins carry nourishment *to* all the body, and that there are two distinct circulations, arterial and venous, which only intercommunicate by hypothetical anastomoses in order that they may participate in each other's qualities. Further, he believes that the nerves are in direct communication with the arteries. He combines the Galenic ebb and flow of the blood with the Aristotelian modification that relegates it to the periods of sleeping and waking. It is not surprising, then, that the writings of Cæsalpinus had little effect upon his contemporaries.

Hieronymus Fabricius, 1537.

“De Venarum Ostiolis,” p. 1 (“Ostiolorum Usus”).

An interesting example of the survival of Galenic influence even after the time of Vesalius is that of Fabricius, who was appointed to the chair of anatomy at Padua in 1565. In “De Venarum Ostiolis,” 1574, he describes the valves in the veins with beautiful illustrations. He regarded them as serving a double purpose: first, the prevention of a too rapid flow of venous blood from the heart to the tissues, in order to give the tissues an opportunity to appropriate the kind of nutriment they require; and second, the prevention of an accumulation of blood in the dependent parts. So by his blind adherence to the Galenic doctrine of crude blood passing from heart to body by the veins he missed the logical conclusions of his own discovery. Fabricius was certainly the first to describe the valves of the veins in detail, and with illustrations, although they had previously been noticed by Galen, Sylvius, Winter, Vesalius, Eustachius, Paul Sarpi the Venetian, and others. This discovery is of interest to our subject, since the venous tracing taken by the clinical polygraph is dependent upon venous valves.

Bartolommus Eustachius, 1524–1574.

His contemporary Eustachius first described the fold of membrane at the opening of the inferior vena cava into the right auricle.

WILLIAM HARVEY AND THE DISCOVERY OF THE CIRCULATION.

In this short historical study we now come to the outstanding figure—William Harvey, the discoverer of the circulation.

William Harvey, 1578–1657.

In 1597 he graduated in arts at Cambridge, and then studied four years under Fabricius at Padua, and thence drew that inspiration towards the study of the heart which would come so directly from that school and the influence of Vesalius and his successors. There he saw Fabricius demonstrate the valves of the veins, and may even have become acquainted with the observations made by Cæsalpinus of Pisa, though he does not refer to him. He returned to England in 1602, and was appointed physician to St. Bartholomew's Hospital in 1609. In 1615 we find him appointed as Lumleian Lecturer, and in 1616 describing his investigations into the circulation and giving his conclusions, though it was not until 1628 that they were published in his great work, "De Motu Cordis." In his notes for the Visceral Lecture in 1616 we find—"It is plain from the structure of the heart that the blood is passed continuously through the lungs

"Life of Harvey," by D'Arcy Power.

to the aorta as by the two clacks of a water bellows to raise water.

"It is shown by the application of a ligature that the passage of the blood is from the arteries into the veins. Whence it follows that the movement of the blood is constantly in a circle, and is brought about by the beat of the heart. It is a question, therefore, whether this is for the sake of nourishment, or rather for the preservation of the blood and limbs by the communication of heat, the blood cooled by warming the limbs being in turn warmed by the heart."

In order to realise what an enormous stride Harvey had made in the journey of discovery, it is well to remember what were the prevalent beliefs of his day. Though the Galenic theory of a permeable septum had been overthrown by the leading spirits of the last century, and the pulmonary circulation was understood to this extent, at least, that the blood which entered the right side of the heart was known to pass to the left through the lungs; yet it was thought that only a portion of the blood went through the heart, the greater part surging to and fro along the two entirely different and practically disconnected systems—arterial and venous. They supposed that the object of such communications as they recognised was not to transfer the blood as a whole from the arteries to the veins, but to allow of the arteries receiving nourishment and the veins spirit. It was a decided step from this belief to William Harvey's pronouncement of a complete pulmonary circulation, in which not only a part but the whole venous blood passed through the heart, from the right side to the left by way of the lungs.

See also De Motu Cordis," chap. ii

Equally important was his description of the propulsive action of the heart, as first described in the Lumleian

Lectures, when systole was substituted for diastole as the primary agent in the cardiac cycle.

But farther-reaching than all was his revolutionary discovery of the systemic circulation. It is extremely interesting to notice the quantitative process, in conjunction with "repeated vivisections" (upon which the opening chapters of his book insist), by which Harvey arrived at the truth that had so long eluded the observation of others. This success was the result of his having "frequently and seriously" considered, and "long revolved in his mind," the question of the quantity of blood transmitted, and, as he says, he found it "not possible that this could be supplied by the juices of the intestinal aliment without the veins on the one hand becoming drained, and the arteries on the other becoming ruptured through the excessive charge of blood, unless the blood should somehow find its way from the arteries to the veins, and so return to the right side of the heart. I began to think whether there might not be a motion as it were in a circle." He describes his conclusion that the blood, "forced by the action of the left ventricle into the arteries, is distributed to the body at large, and its several parts (in the same manner as it is sent through the lungs, impelled by the right ventricle into the pulmonary artery), and that it is then passed through the veins and along the vena cava, and so round to the left ventricle in the manner aforesaid. Which motion we may be allowed to call circular." Once this point was reached, he advanced rapidly, stride upon stride, to further discovery, and fact upon fact was added in proof of this theory of the circulation. One example of this was his demonstration that when a moderately tight ligature is applied, constricting the vein only, the limb beyond becomes swollen, whereas a tighter ligature, constricting the artery, leaves

"De Motu Cordis," chap. viii., p. 41, Francofurti, 1628.

the limb pale and bloodless. Another example was the process of drawing the whole blood of the body by opening one of the larger veins, but he says: "I have never succeeded in tracing any connection between arteries and veins by a direct anastomosis of their orifices." This is not surprising, as Harvey had only a magnifying lens to work with, the microscope not having been yet invented. He seems to have thought that the arteries ended in open mouths in the areolæ of the tissues which they supplied, and that the blood was carried away by the veins in the same manner. As a result of these discoveries he was led to a true explanation of the function of the valves in the veins, so entirely misunderstood by his master, Fabricius, despite his knowledge of their structure. Harvey says most emphatically that it was not through knowing of the existence of valves in the veins that he learnt the truth about the circulation, but, on the contrary, his understanding of the circulation led him to a correct interpretation of the function of these valves.

He did not greatly concern himself with the vexed question as to the infusion of blood with natural, vital, and animal spirits, so prevalent even in his day. He merely remarks: "Whether or no the heart contributes anything else to the blood, besides the transposition, local motion, and distribution of it, we must enquire afterwards, and collect out of other observations. Let this suffice for the present, that it is sufficiently evidenced, that in the beating of the heart the blood is transfused and drawn out of the veins into the arteries, through the ventricles of the heart, and so distributed into the whole body." In chapters viii. and xv. he discusses spirit, blood refrigerated and coagulated, and heat.

"De Motu Cordis," chap. v., and see "The Anatomical Exercises of Dr. William Harvey," by Zachariah Wood, 1653.

In describing the autonomy of the heart-beat, some claim that Harvey anticipated Haller as the originator of the myogenic theory; for Harvey described the heart as a muscular force pump; though this opinion was soon qualified by the neurogenic theory of Borelli.

Harvey was not quite free from the medieval combination of mysticism and medical science when he decided that the "innate heat" of the blood is celestial in nature, and "identical with the essence of the stars," and this he determines is the final cause of the motion of the heart and blood. Sir Clifford Allbutt thinks that this Aristotelian theory may have resulted from his reading of Cicero.

"Science and Mediæval Thought," by Clifford Allbutt.

Harvey, like his great forerunner Vesalius, was a courtier as well as a physician, and he attended Charles I. and his family through their many vicissitudes in the Civil War. Undeterred by the distractions of these great events, during wanderings and battles he calmly pursued his studies and investigations whenever it was possible. He achieved a considerable amount of work during the siege of Oxford from 1642 to 1645, and during that time he was appointed Warden of Merton College in place of Sir Nathaniel Brent, a Parliamentarian. Before this we find him, even during the Battle of Edgehill, when in charge of the little princes, sitting under a hedge, engrossed in a book, until aroused by the fall of a bullet from a great gun; after which he threw himself energetically into tending the wounded. We may conclude with his now famous passage from "De Motu Cordis": "To give in brief my view of the circulation of the blood, and to propose it for general adoption, since all things, both argument and ocular demonstration, show that the blood passes through the lungs and heart by the action of the ventricles, and is sent by distribution to all parts of the body, where it makes its way into the veins and pores of the flesh, and then flows by the veins from the circum-

"De Motu Cordis," chap. xiv., p. 58.

ference on every side to the centre from the lesser to the greater veins, and is by them finally discharged into the vena cava, and right auricle of the heart, and this in such a quantity or in such a flux and reflux, thither by the arteries, hither by the veins, as cannot possibly be supplied by the ingesta, and is much greater than can be required for mere purposes of nutrition; it is absolutely necessary to conclude that the blood in the animal's body is impelled in a circle, and is in a state of ceaseless motion; and this is the act or function which the heart performs by means of its pulse, and that is the sole and only end of the motion and contraction of the heart."

Harvey, at the advanced age of seventy-three years, experimentally proved the passage of blood through the lungs, and the impermeability of the intraventricular septum.

He describes his experiment in a letter to Paul Slegel dated London, March 26, 1651, in these words:

"Works of Harvey," by R. Willis, M.D., 1847.

"Having tied the pulmonary artery, the pulmonary veins, and the aorta in the body of a man who had been hanged, and then opened the left ventricle of the heart, we passed a tube through the vena cava into the right ventricle of the heart, and having at the same time attached an ox's bladder to the tube, we filled it nearly full of warm water, and forcibly injected the fluid into the heart, so that a greater part of a pound of fluid was injected into the right auricle and ventricle.

"The result was that the right ventricle and auricle were enormously distended, but not a drop of water or of blood made its escape through the orifice in the left ventricle. The ligatures having been undone, the same tube was passed into the pulmonary artery, and a tight ligature having been put round it to prevent any reflux into the right ventricle, the water in the bladder was now pushed towards the lungs, upon which a torrent of the fluid, mixed with a quantity of blood, immediately gushed forth from the perforation in the left ventricle, so that a quantity of water, equal to that which was passed from the bladder into the lungs at each effort, instantly escaped by the perforation mentioned."

In spite of a somewhat troubled life, Harvey had the satisfaction of living to the age of seventy-nine, long enough to see opposition vanquished, and it was but four years later that his theories were confirmed by the discovery of the capillary circulation. This was effected by Malpighi, who, with the aid of a microscope, demonstrated the capillary circulation in the mesentery of a frog.

Marcello Malpighi, 1628–1694.

Marcello Malpighi, of Bologna, described his discovery in the second of two letters, "Epistola de Pulmonibus," addressed to Borelli in 1661. The capillary circulation, as seen in fishes, was more fully described by Leeuwenhoeck in 1668.

Leeuwenhoeck, 1632–1723. "Den Waar Omloop des Bloods," 1688.

The work of Harvey appears to have had no influence upon his contemporary, Von Helmont, founder of the iatrochemical school. The great chemist described six digestions in the body; although in the sixth he rightly asserted that the tissues are dependent upon the blood for their life, yet he was a Galenist in his account of the fourth and fifth digestions—*i.e.*, in the heart and arteries. He believed in the permeability of the septum, and had an original notion that vital spirit, being thinner than blood, and aided by the conical shape and disposition of the pores in the septum, passed from the left ventricle against the blood-stream into the right, and there began to operate upon crude blood to convert it into vital blood.

Von Helmont, 1577–1644.

"Ortus Medicinæ," 1648. Sex. Digest., pp. 208–225; and "Spiritus Vitæ," pp. 196-201.

Harvey's theory of blood being expelled from the ventricle during the systolic period was not universally

Ex-adv. J. Harv., 1630. Primerose. Refut. Exerc. Harv., 1639. Parisanus, "Digressio ad circ. sanguinis," 1647. Hoffmann, **Rene Descartes,** 1596–1650. "De Homine," 1662, par. 6, 7, 8.

accepted, his chief opponents being Primerose, Parisanus, and Caspar Hoffmann. We find Descartes, who was a supporter of the main principles of Harvey's circulation, ascribing the pulse to an extraordinary rarefying process in the blood. He believed that each drop of blood, on entering either ventricle, immediately became rarefied, and so greatly enlarged that it produced intraventricular pressure sufficient to force open the aortic valves and so to pass blood into the arteries as to cause the pulse. The rarefied blood remaining in the ventricle then condensed, and the heart and arteries returned to their normal size.

Harvey's discoveries opened the way to so much physiological discovery in connection with the heart that, in dealing with the work of his successors, it will be necessary to confine ourselves more closely to their advancement of knowledge of the heart-beat, and to omit any detailed reference to their general discoveries outside the subject. It must, however, be remarked that such work as that of Malpighi, who laid the foundation of histology, was at the base of all subsequent physiological research. Besides, it is not within the scope of this book to enter into the pathology of the heart; hence the omission of so many great names familiar to all clinicians. It will be necessary, however, in connection with the gradual advance of the science of the heart-beat, to include some discussion of muscle function.

The physiology of muscle was partly understood by Vesalius, for in the "De Fabrica" he recounts how by cutting or ligaturing a nerve he was able to throw the subsidiary muscle out of action, and by relaxing the ligature to bring the muscle into use again.

Up to this time it was generally supposed that skeletal muscular contraction involved not the fleshy part, the so-called "carso," but the tendinous fibres, and that it was effected by the passage of animal spirits along the nerves entering the muscle. Stenson, on the contrary, rightly described the "carso" or fleshy part as the subject of contraction, and not the tendinous fibres. He also discovered that the contraction of muscle is that of its individual parts, arguing that, if a long muscle be cut up longitudinally into several bits, each bit can be made to contract, and hence that the power of contraction lies in the integral substance of the muscle as substance and not (as Borelli declared) entirely in the muscle as a whole.

Niels Stenson, 1638–1686.

"De Musculis Observationum Specimen," 1664.

Stenson also stated that muscular contraction can take place independently of arteries, veins, and nerves.

Considering that it was written as early as 1664, the following passage is remarkable as showing how near to modern lines was Stenson's enquiry into muscular contraction: "There remains another difficulty of no less moment not yet cleared up—namely, in what respect the movement of the fluid in a muscle while it is contracting differs from the movement of the fluid in the same muscle when it remains quiet, uncontracted. Is its quantity changed? Or does it remain the same? Supposing the fluid does remain after the event, is it then the same as before? Does the fluid move because the muscle contracts; or does the contraction of the solid proceed from the movement of the fluid?" His colleague, Thomas Willis, gives a most vivid description of muscular contractions, in which he ascribes them to the action of animal spirits, formed in the ventricles of the brain, which pass down the nerve into the tendinous fibres of the muscle So active and elastic are these spirits that, when opportunity arises, they leap into the fleshy part of the muscle and so cause it to contract.

"Steno. Element. Myol. Spec.," 1667, p. 64.

"Opera Omnia," 1682; "De Motu Musculari," p. 32.

Borelli of Pisa, Malpighi's colleague, spent his life

Giovanni Alfonso Borellij, 1608–1679.

in an attempt to explain the human organism from a mechanical point of view. His views were expounded in "De Motu Animalium," published one year after his death. He accepted Harvey's circulation and Stenson's hypothesis of contraction so far as it concerned the fleshy part of the muscle. He noticed that during contraction the cardiac chambers diminish in size, but explained this by suggesting an increase in the muscle substance during contraction.

"De Motu Animalium," ii. 5, prop. 66, p. 130.

"De Motu Animalium," 1680, ii., prop. 24, p. 59.

On the assumption that all healthy muscle has equal contraction power per unit volume, he attempted to estimate the force of the cardiac contraction by the weight a man could support from his lower jaw with his mouth closed, assuming that the bulk of both ventricles is equal to that of a temporal and masseter muscle. He likened a nerve to a rod of elder pith filled with a fluid "succus nerveus," which he substituted for the Galenic animal spirits. His notion of contraction was that this "succus nerveus" passes from the nerves into the muscles, and there starts a fermentation which produces an actual increase in the volume of the muscles; hence he may be regarded as the founder of the neurogenic theory of cardiac contractions.

"De Motu Anim.," ii. 6, prop. 79, p. 157.

In discussing the origin of the heart-beat he suggests: "It may arise by organic necessity, the heart may move as certain automata move; or possibly the movement may come from a voluntary effort of which we have ceased to be conscious because it has been repeated so often and so constantly." The latter alternative was probably suggested by his recognition that the movement of a limb is due to a direction of the will.

In spite of the valuable work of Descartes, Borelli, and Sanctorius, representatives of the iatromathematical school, in which mathematical laws were rigidly applied to physiological occurrences, we find that the labours of their successors deteriorated to such eccentricities as Edward Barry's attempted calculation of a man's age from his pulse-rate or Clifford Wintringham's futile effort to find the weight of an individual spermatozoon.

"Enq. Ident. Vess. Hum. Body," Wintringham, 1743.

John Mayow, 1643–1679.

Although the name of John Mayow will always be, associated primarily with the discovery of respiration it must be noticed that he had something to say about the heart, and applied his "spiritus nitro-aereus" to muscular contraction. According to Mayow, contraction is due to the fermentation caused by the combination in the muscle of combustible sulphurous particles, and the spiritus nitro-aereus, which is borne to the muscle by the nerve. This view is somewhat contradictory to the following passage on cardiac contraction, in which he says: "If in the heart of an animal just dead, filled not with blood but with water, or some simple liquid, you excite a movement like that which takes place in systole, the contents of the ventricle are forthwith ejected. Not, indeed, by reason of fermentative explosion, for such in this case cannot certainly take place, but simply because the ventricles are contracted." By his work on respiration he was convinced that this function is not to cool the heart and blood, for, as he very poignantly remarks, if that were the case, the rate of breathing during a fever would be much greater than it is during a brief period of great exertion.

Tract. Quinque, 1674, "De Sal Spiritu et Nitro-aereo."

Ibid., "De Motu Musc., 4, p. 29.

Ibid., "De Respiratione."

Francis Glisson, 1597–1677.

It was left, however, to an Englishman, Francis Glisson, to refute these false notions of muscular contraction. His opinions had little influence upon his own time, but were revived by Haller in the middle of the eighteenth century. In Glisson's book he introduces into physiology the term irritability. In his work "De Ventriculi" he denies

"Tractatus de Natura Substantiæ Energetica," 1672; "De Irritabilite," cap. 7 and 8; "De Ventriculi," cap. 7.

that muscle contracts in response to animal spirits, or to Borelli's succus nerveus; and, more important still, by inventing a plethysmograph, in which he placed a man's arm, he proved that muscle when contracted does not increase in volume. He was wrong in concluding that contracted muscle diminishes in volume, though right in his conclusion that individual fibres become shorter. His mistake in thinking that the volume of muscle is less in contraction than in relaxation was due to his not recognising that during contraction blood is squeezed out of the vessels.

Lancisi, 1655–1720.

Lancisi's work "De Motu Cordis et Aneurysmatibus" (1728) gives a description of the undulations of the cervical veins which is very interesting in connection with our present-day use of the clinical polygraph.

Richard Lower, 1631–1690. "Tract. de Corde," 1669, cap. 3.

Richard Lower attracted attention by his transfusion and infusion experiments. He described the structure of the heart more accurately than had ever been done before. He discussed the function of the heart on the same lines as Harvey; but an increased mathematical knowledge enabled him to give more accurate figures in calculating the amount of work done by the heart. That the crimson colour of arterial blood was not due to any influence exerted on it by the action of the heart was proved by Lower in the following experiment: he showed, after opening the chest of a live dog, that the blood in the pulmonary artery is as dark as that in the vena cava, and that after tying the windpipe the blood from a branch of the aorta is no longer crimson. But when the ligature on the trachea is released, or the lungs inflated by bellows, the blood immediately recovers its crimson colour. He rightly concluded that the bright colour of arterial blood is dependent upon the atmospheric air inspired. From the fact that arteries have thicker walls than the veins he deducted that pressure is greater in the former than in the latter.

James Keill was of the school which applied mathematics to physiology, and so founded animal mechanics. Borelli may be described as the originator of that department of science, but James Keill went farther in the investigation of the force of the heart. In his book he calculated the velocity of blood in the aorta at 52 feet a minute; he then showed that on account of resistance the velocity of blood decreases towards the smaller vessels, until finally in the smallest he estimated that it travels $\frac{1}{4}$ inch in 278 days! He further described the hydraulic phenomena of the circulation in "Essays on Several Parts of Animal Economy," 1717. On a dynamic basis he concluded that the force of the heart is "little above 16 ounces." A great divergence from Borelli's conclusion of 135,000 pounds.

James Keill, 1673-1719.

"Animal Secretion and the Quantity of Blood in the Human Body and Muscular Motion," 1708, Essay III.

It may be asked why Keill went so far wrong in his ingenious attempt at quantitative estimates? The answer in his case, as in Borelli's, lies in his inaccurate data, for they both paid so much attention to mathematical calculation that they overlooked the necessity of practical experiment and careful observation. These were supplied by Stephen Hales, Rector of Teddington, to whom credit should be given for the first substantial advance in the physiology of the circulation since the time of Harvey. He was a man of great inventive genius, and is popularly known as the originator of artificial ventilation. In his "Statical Essays" he describes his famous experiment in the determination of blood-pressure as follows: "In December I caused a mare to be tied down alive on her back. . . . Having laid open the left crural artery about 3 inches from her belly, I inserted into it a brass pipe whose bore was $\frac{1}{6}$ inch in diameter,

Stephen Hales, 1677-1761.

"Statical Essays—Hæmostatics," vol. 2 p. 1. 1733.

and to that, by means of another brass pipe which was fitly adapted to it, I fixed a glass tube of nearly the same diameter which was 9 feet in length; then untying the ligature on the artery the blood rose in the tube 8 feet 3 inches, perpendicular above the level of the left ventricle of the heart; but it did not attain to its full height at once: it rushed up about half-way in an instant, and afterwards gradually at each pulse, 12, 8, 6, 4, 2, and sometimes 1 inch; when it was at its full height, it would rise at and after each pulse 2, 3, or 4 inches; and sometimes it would fall 12 or 14 inches, and have there for a time the same vibrations up and down, at and after each pulse, as it had when it was at its full height, to which it would rise again after forty or fifty pulses."

He then estimated the capacity of the left ventricle by using waxen casts, and calculated that the force of the left ventricle in man is about equal to that of a volume of blood 7½ feet high—*i.e.*, 57½ pounds. If we compare these results with the over and under estimates of Borelli and Keill respectively, we feel justified in calling Hales the founder of modern experimental method in physiology, and we can hardly fail to appreciate his experimental and methodical process as compared to the fanciful calculations of his predecessors.

This subject attracted the attention of physicists such as Jurin and Bernouilli, and also of Haller, Bernouilli's pupil in mathematics.

Albrecht von Haller, 1708–1777.

Albrecht von Haller, who graduated at Leyden, had a very wide knowledge of many subjects, including medicine and botany. He taught anatomy, physiology, and botany, at the newly founded University of Göttingen until 1753, when he retired to Berne. In addition to his interest in the subjects mentioned above, he composed poetry, and was the founder of medical and scientific biography. He is noted in the history of recent physiology for his laboratory demonstrations elaborating Glisson's theory of irritability; these were based on 567 experiments made at Göttingen, of which 190 were performed by himself, and they proved that irritability is a specific property of muscle. He contrasted this property, "vis insita," with the somewhat similar "vis nervosa," which, however, was not inherent in muscle, but reached it by the nerve from the brain. In discriminating between the specific irritability of muscular tissue and that of nerves and other tissues he refers to that property of irritability in muscle which makes itself manifest by movement. This function of irritability he applied to heart-muscle, and so founded the myogenic theory of cardiac contraction. Of Haller's book "Elementa Physiologiæ Corporis Humani," Sir Michael Foster says, "to open this is to pass into modern times," for therein will be found many so-called "new discoveries" of modern observers, really made by Haller but passed over and forgotten by his successors.

"Comment. Soc. reg. Gottingæ," ii. 114–214, 1753.
"Elementa Physiologia," 1762, lib. xi., p. 460.
Ibid., lib. xi., p. 446.
Ibid., lib. xi., p. 468.

"Elementa physiologiæ corporis humani," Lausanne, 1759–1766.

Haller died with his finger on his pulse, and his last words were: "The artery no longer beats."

Leopold Auenbrugger was the first to discover the clinical application of percussion.

Leopold Auenbrugger, 1722–1809.

The result of his seven laborious years of research are recorded in his little book, "A New Invention for Discovering Obscure Thoracic Diseases by Percussion of the Chest" (1761). This important mode of examination was slighted at the time, but revived in 1808 by Corvisart, who unearthed Auenbrugger's book and published it in 1811, declaring that he could thus estimate the diameter and circumference of the heart. There is some evidence, even in the works of Hippocrates and his successors, that percussion as a clinical method was not unknown,

Jean Nicolas Corvisart, 1755–1821

but it had no real value until it was brought forward by Auenbrugger and Corvisart.

Lænnec, 1731–1826.

Following upon this was the further discovery of yet another clinical method of examination, the stethoscope, invented by Lænnec in 1819. This instrument in his original experiment consisted of a paper cylinder only, and is described in his work "Traité de l'Auscultation Médiate," published in two editions, 1819 and 1823. In the importance of his discovery Lænnec should rank with Vesalius and Harvey. He died of phthisis at the early age of forty-five.

Thomas Young, 1773–1829.

Thomas Young investigated the effects of friction, etc., in the circulation, and some phenomena of pulsation, in a paper entitled "Hydraulic Investigation." This was followed by his Croonian Lecture, in 1808, called "On Functions of the Heart and Arteries," in which he clearly demonstrated the following principles: that there is a decrease in the velocity of blood as it passes from the larger to the smaller vessels; that the resistance it encounters is chiefly in the smaller vessels; and that the elasticity of the walls of the large arteries maintains during diastole a continuous flow of blood against the resistance of the small arteries. He also showed that the function of the muscular coats of the smaller arteries was not to propel, but to regulate the blood-supply.

Poiseuille, 1799–1869.
"Rech. sur la force du cœur aortique," Thèse, Paris, 1828.

The work of Poiseuille was directly based upon the original discovery, by Stephen Hales, of the manometer; but by his invention, in 1828, he improved upon the straight pipe of Hales's cumbersome instrument by substituting a U-shaped tube containing mercury. This was connected with the artery by rubber tubing filled with a solution of sodium bicarbonate to prevent coagulation of the blood.

This instrument was further improved upon by Carl

Ludwig, who, in 1847, added a float, and, as Professor Stirling says, "had the genius to cause the float to write on a recording cylinder, and thus at one coup gave us the kymograph, or wave-writer, and the application of the graphic method in physiology." It is impossible to overestimate the importance of this discovery, for it opened the way to those great strides in experimental physiology, all of which depended upon the power of recording movements exactly to measurement and time.

Carl Ludwig, 1816–1895. *Arch. f. Anat. Physiol. u. Wissensch. Med.*, 1847, S. 261.

One can estimate the enormous importance of this discovery of recording movements, when one remembers the almost insurmountable difficulties which, before its invention, faced the pioneers of research, especially in the timing of muscular contraction. These are illustrated by Harvey's own words in the opening chapter of "De Motu Cordis": "When I first gave my mind to vivisections as a means of discovering the motions and uses of the heart, and sought to discover these from actual inspections, and not from writings of others, I found the task so truly arduous, so full of difficulties, that I was almost tempted to think, with Fracastorius, that the motion of the heart was only to be comprehended by God. For I could neither rightly perceive at first when the systole and when the diastole took place, nor when and where dilatation and contraction occurred, by reason of the rapidity of the motion, which in many animals is accomplished in the twinkling of an eye, coming and going like a flash of lightning, so that the systole presented itself now from this point, now from that, the diastole the same; and then everything was reversed, the motions occurring as it seemed variously and confusedly together; my mind was therefore greatly unsettled, nor did I know what I should myself conclude nor what believe from others."

"The Works of William Harvey," by Robert Willis, M.D., 1847.

The influence of Ludwig's multifarious works spread over the whole field of physiology. Instrumental inventions and further important discoveries became so numerous that it is not possible in this book to refer to many of them, either in detail or in chronological order. I propose to mention a few of the more important types of the instruments which have played so great a part in the advancement of our knowledge of the heart-beat. Many of the discoveries will be mentioned later, in discussions of the functions of cardiac muscle and the nerve-supply of the heart. These I will classify under typical headings, for the sake of future reference.

Journn. Physiol., vols. iii., iv., and vi.

It may be noticed that the method introduced by Ludwig of keeping tissues alive extra-vitally has enabled the physiologist to carry out those far-reaching experiments which it would otherwise have been impossible to perform. We owe the perfection of this process to Sidney Ringer, who demonstrated both the importance of calcium salts for the contraction of muscle and also the necessity of having a definite quantity of sodium and potassium salts in the circulatory fluids.

Before considering the development of instruments and instrumental methods the simple process of pulse-timing should be mentioned.

"The Early History of Instrumental Precision in Medicine," by S. Weir Mitchell, New Haven, 1892, p. 10.

We find pulse-counting employed by Kepler in 1600, as a method of timing astronomic phenomena, and similarly it was used by Galileo, who counted his own pulse in testing the time of a pendulum's vibrations. In Galileo's case, however, this led further to the idea of counting the rate and variations of the pulse by the pendulum very much as music is timed by a metronome.

Sanctorius, 1561–1636.

Sanctorio Sanctorio, or Sanctorius, a Paduan professor, took advantage of these applications and made remarkable use of them. We find him describing a pulsilogium, or pulse clock, which he had himself invented; but for a while these were soon forgotten, and did not come into general use until nearly a hundred years later. Sanctorius founded metabolic physiology concerning what he called the "insensible perspiration" of the body. He appears to have been an inventive genius, for in the later editions of his book, "Ars de Statica Medicina" (1614), there is a frontispiece showing him in one of his experiments in metabolism weighing himself after a meal in a steelyard chair.

"Commentary on the First Book of the Canon of Avicenna," Venice, 1625, column 21, CC.

One of the most interesting efforts to use instruments of precision in diagnosis is that of Sir John Floyer of Staffordshire, who published in 1707 his "Physician's Pulse Watch." That book describes the first attempt to revive exact pulse-timing since the days of Galileo and Sanctorius. It must not be supposed that they were the originators of this practice, for we find the ancient Herophilus of Alexandria counting the pulse with his clepsydra.

John Floyer, 1649–1734.

It should be noticed that the ordinary watch of that period had a single hand only, recording hours, but neither minutes nor seconds; but Floyer was clever enough to invent a watch which ran for exactly one minute, by which the pulse could be timed with some degree of accuracy.

In other branches of medical science, and especially in pharmacy, the old difficulty of having no instrument to record short periods of time was overcome by the ingenious device of repeating certain specified prayers or psalms or verses as a measure of the time which should be allowed to various performances such as the boiling of medical concoctions; but in pulse-timing this method would obviously be impossible.

It is surprising that even Floyer's work was neglected or even misdirected in the revival of the old Galenic doctrines of specific pulses. This period of pulse lore is described by Dr. Weir Mitchell, the historian of instrumental precision in medicine, as "observation going minutely mad, a whole Lilliput of symptoms, an exasperating waste of human intelligence"; and he adds that, "it was not until a later day, and under the influence of the great Dublin school, that the familiar figure of the doctor, watch in hand, came to be commonplace."

Trans. Cong. Am. Phys. and Surg., 1892, ii., p. 179.

Théophile de Bordeu, 1722–1776. "Recherches sur le Pouls," 1756 and 1768.

We should mention Théophile de Bordeu, who founded the vitalistic school of Montpellier for his complex classifications of the pulse as critical, non-critical, simple critical, compound critical, nasal, tracheal, gastric, renal, uterine, seminal, etc.; but his work was purely fanciful.

Louis, 1787–1872. "Généralités sur l'enseignement de la médecine clinique," 1831.

Floyer's method of using a watch to time the pulse was first taken up by Pierre-Charles Alexandre Louis, the founder of medical as separated from vital statistics.

Corrigan, 1802–80.

Edin. Med. and Surg. Journ., 1832, xxxvii. 225-245.

In 1832 Sir Dominic John Corrigan published his classical description of aortic regurgitation, and described the visible pulsation of the arteries now known as the "Water-hammer" or "Corrigan" pulse; and suggested tapping the precordial region with a hot spoon (Corrigan's hammer) to stimulate a flagging heart.

Samml. klin. Vortr., Leipzig, 1873, No. 54, 1637–1674. *Prag. med. Wochenschr.*, 1903, xxviii., 377–381.

We should notice the introduction of the idea of the "pulsus paradoxus" by Adolf Kussmaul in 1873; and Hering's description of the "pulsus irregularis perpetuus," which is now known to be due to auricular fibrillation.

PART II

THE DEVELOPMENT OF INSTRUMENTS AND INSTRUMENTAL METHODS

The following is a description of the more important types of instruments and experimental methods which have led to our present exact knowledge of the heart-beat.

1. Endocardial Blood-Pressure Method.

In 1861 Chauveau and Marey introduced the cardiac sound, which could be passed down the jugular vein into the right auricle or ventricle, or down the carotid artery into the left ventricle. In this way they obtained direct records of the pressure changes in the heart of a horse, and so were enabled to establish the sequence of contraction of the heart chambers. This instrument consisted of a stiff tube, the end passing into the heart being covered by an elastic ball, the other connected with a tambour and writing lever. It was too large to be used on the smaller animals, so that to overcome this difficulty Karl Hürthle introduced his membrane manometer. Hürthle, by combining two of his instruments so that two tambours were connected to the writing lever, one on each side of the fulcrum, invented the differential manometer. He introduced one tube of this instrument into the right auricle, and the other into the right ventricle, and so was enabled to record the exact moment at which the auricular pressure rose to an excess of the ventricular and conversely, and thus he indicated the exact time of the opening and closing

Mém. Acad. de méd., Paris, 1863, tome xxvi., p. 272. "Circulation du Sang," by Marey, Fig. 45, p. 111.

Hürthle, *Arch. f. d. Gas. Physiol.* Bonn, 1891, Bd. xlix., S. 45.

of the tricuspid valves. Similarly the opening and closing of the aortic valves were recorded by placing one tube in the aorta, and the other in the left ventricle.

The disadvantage of Ludwig's manometer was that the inertia of the mercury was so great that those rapid changes of pressure which take place in the heart could not be recorded accurately. The inventions of Chauveau and Marey and of Hürthle were designed to overcome this difficulty, but the writing levers connected with the tambours gave rise to considerable instrumental errors, and the problem was not solved until Piper produced his improved manometer. This consisted of a short tube with a stilette which accurately fitted the tube, and passed through a stopcock, and a short rubber membrane was fixed over a hole in the side of the tube. This instrument was introduced through the wall of the heart or blood-vessel, and secured by a purse-string suture; the stilette was then withdrawn, and the stopcock turned off. The variations of blood-pressure were recorded on a photographic plate by means of a beam of light reflected from a small mirror attached to the rubber membrane.

Arch. Anat. Physiol., 1912, p. 343.

2. SUSPENSION METHOD.

The heart is suspended by a clamp in the auriculo-ventricular groove, tight enough to hold it without interfering with the conduction of stimuli from the auricle to ventricle. Writing levers, working against delicate springs, are attached by threads to the muscles of the auricle and ventricle. This apparatus proved very useful in the hands of Gaskell, who first introduced it.

Phil. Trans., part iii., 1882, by W. H. Gaskell.

3. Perfusion Method.

We have already noted that it was Ludwig who, in 1865, first introduced the method of perfusion for keeping organs and tissues alive outside the body for experimental purposes; and that Sidney Ringer perfected the perfusion fluid by introducing what is now known as Ringer's solution.

Journ. Physiol., vols. iii., iv., and vi.

This method has not only been used for the purpose of keeping the heart or other tissues alive, but more recently it has been employed to examine the effect of different solutions, including soluble gases, upon the action of the heart. I do not propose to give a description of the many methods of perfusion, but merely to mention the two well-known examples—Gotch's frog-heart apparatus and Brodie's perfusion apparatus, sketches of which will be found in any textbook of physiology.

"Principles of Human Physiol.," by E. H. Starling. Ed. 2, p. 964.

Journ. Physiol., 1908, vol. xxxvii., p. 337.

4. Blood-Pressure, Blood Velocity, and Output of the Heart.

It is not intended to discuss these subjects, but merely to enumerate the more important instruments and apparatus employed.

The pioneer in the investigation of blood-pressure was Stephen Hales, whose classical experiments have already been described. The first to attempt to estimate blood-pressure without opening the artery was Vierordt, who in 1855 conceived the idea of measuring the pressure by applying weights over the artery until the pulsation ceased. Working on this principle, Ritter von Basch invented a small clinical sphygmometer, consisting of a rubber balloon filled with water and connected by a rubber tube to a manometer. The balloon was pressed on the artery

"Die Lehre von Arterienpuls," Braunschweig, p. 164, 1885.

Wien. med. Wochenschr., 1883, S. 673.

Ztschr. f. Klin., Berlin, Bd. ii., S. 79.

Berl. klin. Wochenschr., 1887, S. 181.

until pulsation ceased, and the manometer reading then taken.

In 1876 Marey brought forward an instrument which, instead of pressing upon a limited part of an artery, was based upon the principle of constricting the whole limb until pulsation ceased. In 1896 this instrument was improved by Riva-Rocci, and later by C. J. Martin, who has given to us the instrument which is now frequently used. As a result of experiments made in 1905 at Petrograd, Korotkow suggested that the stethoscope might be used to determine arterial blood-pressure in man, and Krylow put the use of it into clinical practice. More recently, J. A. MacWilliam has done some valuable critical work on the subject, and the present-day custom is to take the systolic and diastolic blood-pressure by the auscultatory method, using one of the modern sphygmomanometers. The stethoscope is placed at or below the bend of the elbow, and as the pressure in the armlet is raised in the usual way distinct sounds are heard with each heart-beat. The pressure is raised until the sounds disappear, then air is allowed to escape slowly from the armlet, and the manometer reading is taken at the time the first sound reappears. This is the maximum or systolic blood-pressure. As the air continues to escape the sounds first become clear, then muffled, and finally disappear. The manometer reading at the time at which the sounds suddenly change from being clear to muffled is usually taken as the minimum or diastolic blood-pressure; but a few workers prefer to use the manometer reading taken at the time the sounds completely disappear. The pulse-pressure is the difference between the systolic and diastolic blood-pressures.

Gazz. Medica di Torino, 1896, 1897.
Brit. Med. Journ., 1905, i. 865.
"*Mitteilungen der Kaiserl. Milit.-Mediz. Akad. z.*," *St. Petersburg*, 1905, vol. ii., p. 365.
Verhandlungen des Kongresses für innere Medizin, Wiesbaden, 1907, vol. xxiv., pp. 404 *et seq.*
Heart, vol. v., 1914.

The first to attempt direct measurement of the velocity of the blood in an artery was Volkmann, who introduced into the artery a **U**-shaped glass tube filled with salt solution, of approximately the same bore as the artery, noting the time taken by the blood in passing from one end of the tube to the other. This method was improved upon by Ludwig in his stromuhr produced in 1867. For the purpose of measuring the change of velocity during a single heart-beat Chauveau invented the hæmodromograph, and Cybulski the photohæmatachometer. The former consists of a pendulum of which one end is suspended in an artery, the other being connected with a tambour. This instrument comprises an application of Pitot's tubes, one tube being placed directly over the direction of the blood-flow. The two lateral tubes are connected with a manometer whose movements are photographed; the difference in level of the two limbs of the manometer is proportional to the velocity of blood. These instruments must first be calibrated.

"Die Hamodynamik," Leipzig, 1850, S. 185.

Dogiel, *Ber. d. k. Sächs. Gesellsch. d. Wissensch.*, Leipzig, 1867, Bd. xx., S. 200.

Journ. de physiol. de l'homme, Paris, 1869, tome iii., p. 695.

Pflüger's Arch., 1885, vol. xxxvii., p. 382.

The output of the heart during each beat has been directly measured by Knowlton and Starling's apparatus, and by various plethysmographic methods—*e.g.*, those of Roy, Gaskell, and Henderson.

Journ. Physiol, vol. xliv., 1912 p. 206.

It can be measured indirectly in man by Zuntz's method, which is based upon measuring the amount of oxygen taken into the lungs per minute, and estimating the excess of oxygen in arterial blood over that in venous blood. By this method the output of each ventricle in man was found to vary from 50 to 100 c.c. and the average output per beat may be taken as 60 c.c. Krogh's method of estimating the output of the heart at each beat or per minute is based upon the determination of the quantity of nitrous oxide taken up from the mixture of nitrous oxide and air with which the lungs are filled, and thus from a calculation the quantity of blood flowing

Deutsche med. Wochenschr., Leipzig, 1892, S. 109.

"Principles of Human Physiology," by E. H. Starling, Ed. 2, p. 913.

through the lungs in a given time. Krogh finds that the ventricular output in man may vary from 3 to 21 litres of blood per minute.

5. SPHYGMOGRAPHS.

"Die Lehre vom Arterienpuls," Braunschweig, 1855, S. 21.
Journ. de physiol. de l'homme, Paris, 1860, tome iii., p. 241.

Vierordt first applied the graphic method to the investigation of the pulse, but the first sphygmograph accurately recording the form of the pulse and used for clinical purposes was that of Etienne-Jules Marey (1860). This was improved upon by the introduction of Dudgeon's sphygmograph, which could be more easily handled and adjusted.

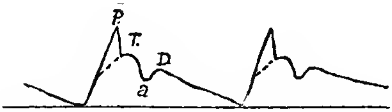

FIG. 2.—DIAGRAMMATIC REPRESENTATION OF THE PERCUSSION, TIDAL, AND DICROTIC WAVES.

Mém. Acad. de méd., Paris, 1863, tome xxvi., p. 284.
Skandin. Arch. f. Physiol., Leipzig, 1889–1891, Bd. i., S. 74.

One of the earliest instruments for recording the apex-beat was Chauveau and Marey's cardiograph, which later was improved by Edgren and others.

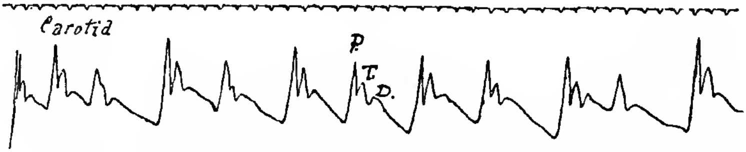

FIG. 3.—CAROTID TRACING SHOWING THE PERCUSSION, TIDAL, AND DICROTIC WAVES.

Fig. 2 represents diagrammatically the percussion, tidal, and dicrotic waves which may occur in a sphygmographic tracing of the arterial pulse.

The percussion wave P is caused by the lever of the instrument being thrown up with a jerk; it falls down on to the true pressure or tidal wave T. If it were not for the instrumental error causing the percussion wave, the tidal wave would take the course of the dotted line. The dicrotic wave D is probably caused by the closure of the semilunar valves; *a* is the aortic notch.

Fig 3 is a tracing from the carotid pulse illustrating the P, T, and D waves.

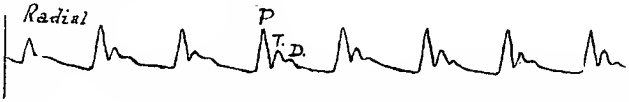

FIG. 4.—RADIAL TRACING SHOWING THE PERCUSSION, TIDAL, AND DICROTIC WAVES.

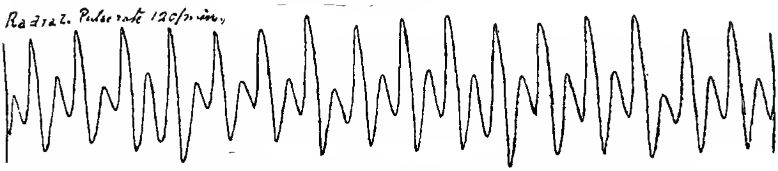

FIG. 5.—RADIAL TRACING SHOWING THE DICROTIC PULSE. TAKEN FROM A CASE OF ACUTE LOBAR PNEUMONIA.

Fig. 4 represents the same waves in a radial tracing.

Fig. 5 is a radial tracing illustrating the dicrotic pulse. This tracing was taken from a case of pneumonia.

6. THE CLINICAL POLYGRAPH.

We have already noted that Lancisi observed the undulations of the cervical veins early in the eighteenth century. Bamberger and Marey graphically recorded the movements of the jugular vein in 1863. The graphic registration and interpretation of the venous and arterial pulses was greatly advanced by the researches of Potain. In 1892, Mackenzie used the venous pulse to interpret irregularities of the heart, and he described the result of his work in two papers in 1893-94. He published "The Study of the Pulse" in 1902. The name of Mackenzie

"De Motu Cordis et Aneurysmatibus," Naples, 1738.

Physiol. Médic. de la Circ. du Sang, Paris, 1863. *Mém d. l. soc. Méd. d. H. d Paris*, 1867, (mai), 3, 27.

Journ. Path and Bact., 1893, i., 53-89.

Ibid., 1894, ii., 84-154.

will always be associated with the interpretation of the venous pulse and the classification of cardiac irregularities. It was he who made such valuable clinical use of the principle that changes of blood-pressure in the right auricle are reflected in the termination of the internal jugular and subclavian veins, since there are no valves between the right auricle and the termination of these two veins. The valves are situated about 1 inch from where these two veins unite to form the innominate vein—see Fig. 9.

By placing a receiver connected with a tambour over this position at the root of the neck—*i.e.*, just above the clavicle, 1 inch external to its articulation with the sternum—the variations of blood-pressure within the right auricle can be recorded.

Mackenzie worked at first with a Dudgeon's sphygmograph, to which he fitted a time marker and recording tambour which was connected by rubber tubing to a small cup receiver. Later he introduced the ink polygraph and first described it in 1908. It is not my purpose to describe this polygraph, since it is now well known and in everyday use. Suffice it to say that there are three tracings recorded at the same time:

Brit. Med. Journ., 1908, i., 1411.

1. A tracing of the arterial pulse—usually the radial artery. This tracing is dependent upon the contraction of the left ventricle.

2. A tracing of variations in venous pressure in the internal jugular vein. This tracing is dependent upon the blood-pressure in the internal jugular vein, which is influenced by the action of the auricles, ventricles, and respiration, and by the position of the individual. The possibility of obtaining a true venous tracing from the neck at any one time is dependent upon the quantity of blood in the jugular vein, the amount of fat at the root of the neck, and other individual peculiarities. If the

quantity of blood is small, no venous tracing will be obtained.

3. A time recorder, each division being usually $\frac{1}{5}$ second.

It is by the aid of the clinical polygraph that Mackenzie unravelled the then tangled skein of cardiac irregularities. His great work was accomplished whilst he was in general practice, a fact which should give inspiration to others so engaged; and so noteworthy were his achievements that they should always rank with those of the past great masters of medicine.

The following are the features shown by a polygraph tracing in normal hearts:

1. The rate at which the heart is beating.
2. That it is beating regularly, both in rhythm (except in sinus arrhythmia) and force.
3. That the auricle and ventricle are beating in a regular sequence.
4. The amount of time taken by the impulse to travel from auricle to ventricle along the auriculo-ventricular bundle.
5. What proportion of time in every beat is taken up by the contraction of the auricles and ventricles and what by their diastole

FACTORS IN THE PRODUCTION OF THE VENOUS CURVE.

Before polygraphic tracings can be interpreted, the mechanism of the cardiac cycle, and its influence upon venous pressure, must be studied. The auricle must be looked upon as a sort of concertina, which by contracting draws up the auriculo-ventricular fibrous ring and empties itself more or less entirely into the ventricle. This movement is effected by contraction of the musculi pectinati, whose fibres take origin in the tænia terminalis and are inserted into the fibrous ring at the junction of auricle and ventricle (see Fig. 6).

When the ventricle contracts there are two fixed points

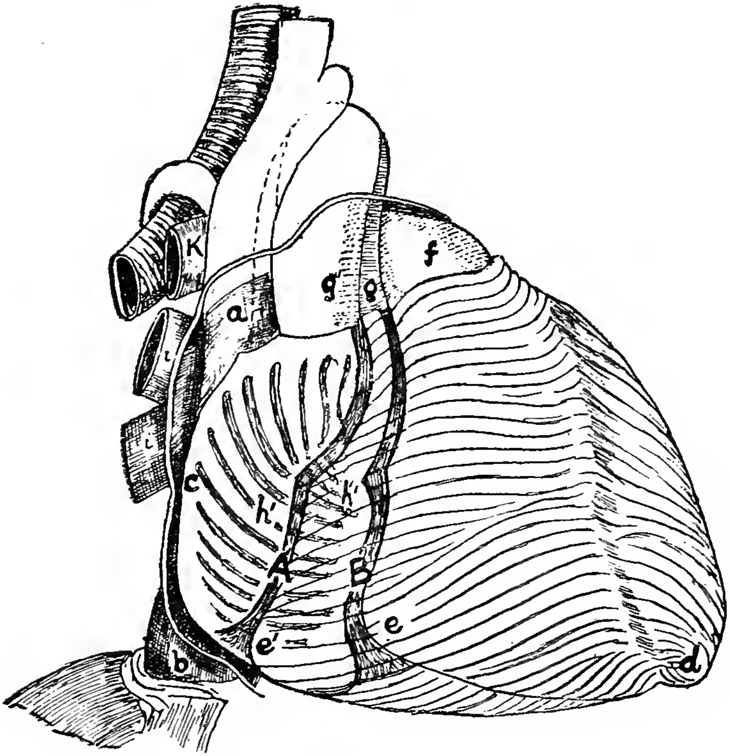

Fig. 6.—Sketch of the Heart to show the Movements of the Auriculo-Ventricular Groove during Auricular and Ventricular Systole.

A. Position of groove when pulled upon by the contraction of the pectinate fibres of the auricle;

B. Position of the groove when pulled upon by the contraction of the ventricular fibres. During the diastole the groove occupies a position midway between A and B; *a*, sinus portion of superior vena cava; *b*, inferior vena cava; *c* is on the tænia terminalis; *d*, apex of the heart; *f*, pulmonary artery; *i*, *i*, pulmonary veins; *k*, pulmonary artery. During ventricular systole *g'* is pulled to *g* and *e'* to *e*; *h'*, musculi pectinati during auricular systole; *h''*, during ventricular systole. (Keith.)

of the heart: the base, which is secured by the fixation of the large vessels, and the apex, which is fixed by virtue of the peculiar arrangement of the musculature constituting the whorl. The diminution of the ventricular cavity during contraction must take place by the ventricular wall moving towards the line joining these two fixed points.

During ventricular contraction, then, the auriculo-ventricular fibrous ring is pulled down. The importance of this movement is pointed out by Keith; not only does it diminish the size of the ventricular chamber, but at the same time it enlarges the capacity of the auricle; this again influences the tracing from the neck by causing a fall of venous pressure in the great veins. The up-and-down movement of the auriculo-ventricular fibrous ring is shown in Fig. 6.

Fig. 7 shows a normal venous tracing and represents the varying pressure in the internal jugular vein.

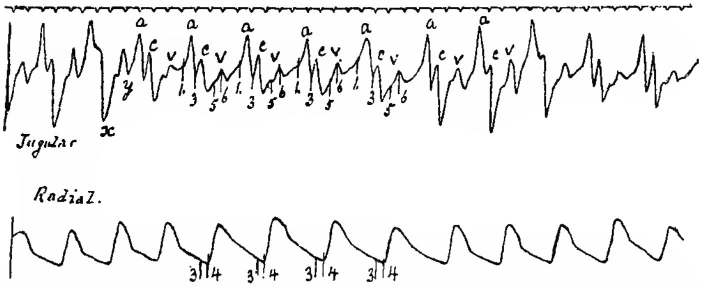

FIG. 7.—SIMULTANEOUS TRACINGS OF THE JUGULAR AND RADIAL PULSES, SHOWING THE STANDARD POINTS OF MEASUREMENT, 1, 3, 4, 5 AND 6, THE AURICULAR WAVE *a*, THE CAROTID WAVE *c*, THE VENTRICULAR WAVE *v*, AND THE FALLS *x* AND *y*.

The wave *a* is due to the contraction of the right auricle; *v* is due to blood collecting in the auricle and great veins during the systole of the ventricle.

The wave *c* is believed by Mackenzie to be arterial and caused by the impulse of the carotid artery, and probably of the subclavian artery also, which is superimposed upon the true venous tracing. Physiologists do not agree with Mackenzie that *c* is arterial, as Straub has demonstrated it in intra-auricular pressure curves, and Starling in tracings taken from the inferior vena cava. Fig. 8 is taken from one of Straub's tracings.

"Principles of Human Physiology,' Starling, Ed. ii., 1915.

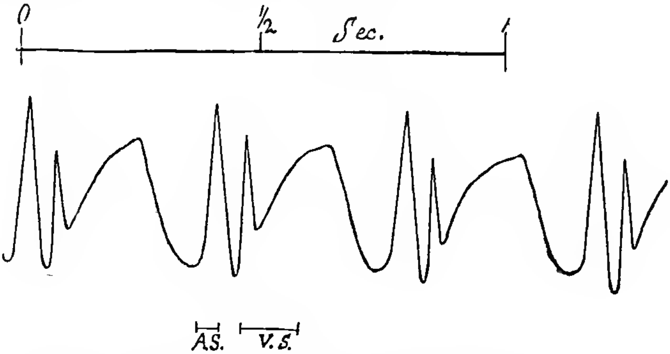

FIG. 8.—CURVE OF PRESSURES IN LEFT AURICLE OF A CAT.

Referring again to Fig. 7, the fall *x*, which represents a fall in the venous pressure, is produced by the three following factors:

1. The relaxation of the right auricle.

2. The expansion of the right auricular cavity, owing to the contraction of the right ventricle pulling down the auriculo-ventricular junction.

3. The diminished intra-thoracic pressure caused by the expulsion of blood from the thoracic cavity in consequence of the contraction of the left ventricle.

The fall *y* is caused by blood passing from the right auricle into the right ventricle at the opening of the tricuspid valve.

These inferences will be appreciated by referring to Fig. 9, which shows the relation of the heart and great vessels; the position of the polygraphic receiver above

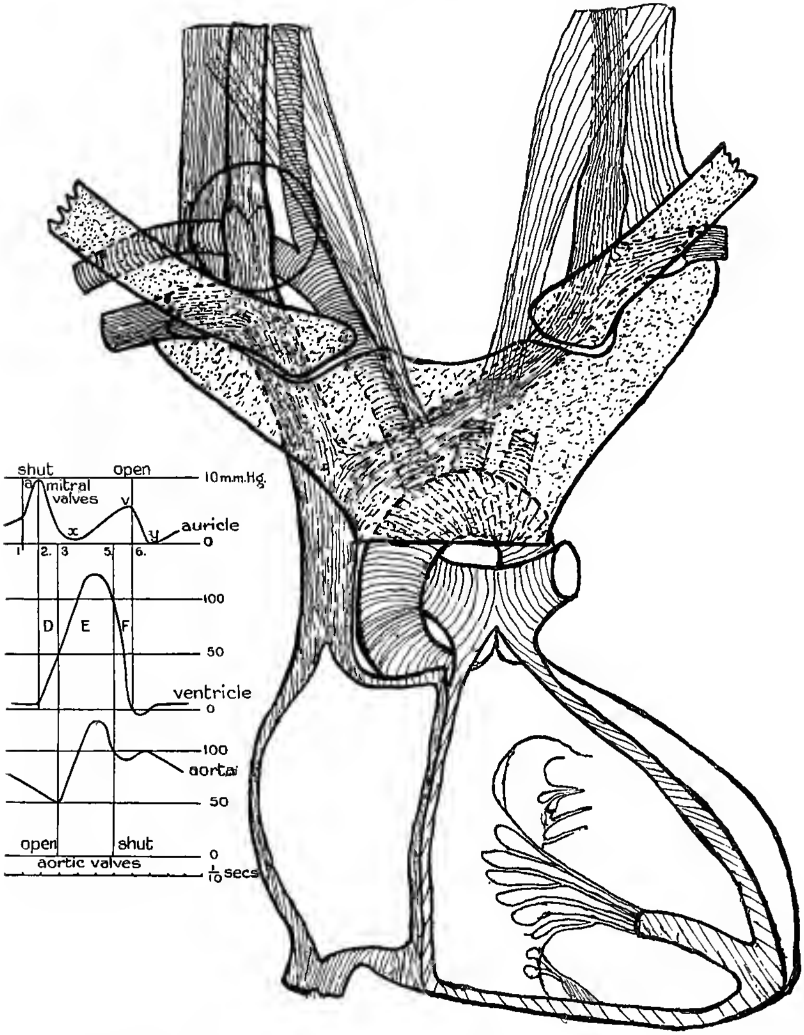

FIG. 9.—A DIAGRAM TO SHOW THE RELATION OF THE HEART AND GREAT VESSELS; THE POSITION OF THE POLYGRAPHIC RECEIVER ABOVE THE CLAVICLE; AURICULAR, VENTRICULAR AND AORTIC PRESSURE CURVES.

the clavicle; and the auricular, ventricular, and aortic pressure curves.

That variety of venous pulse which I have just described is called the " auricular " venous pulse. There is another kind called " ventricular " venous pulse. In this variety the auricle either does not contract or its contraction is not strong enough to affect the pressure in the veins. There are two varieties of the ventricular venous pulse; one shows a single large *v* wave, of which Fig. 10 is an example.

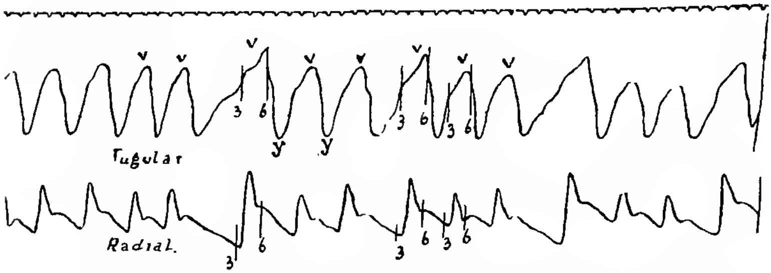

FIG. 10.—SIMULTANEOUS TRACINGS OF JUGULAR AND RADIAL PULSES, SHOWING ONE LARGE WAVE *v*, SYNCHRONOUS WITH AND DUE TO THE VENTRICULAR SYSTOLE; AND ONE LARGE FALL *y*, SYNCHRONOUS WITH AND DUE TO THE VENTRICULAR DIASTOLE.

There is no sign of an auricular wave, and the jugular pulse is therefore of the ventricular type, and the rhythm of the heart is continuously irregular, due to auricular fibrillation.

From Mackenzie's " Diseases of the Heart." London: Oxford Medical Publications.

The other shows a mid-systolic fall of which Figs. 54, 55, and 56, are examples.

THE INTERPRETATION OF VENOUS TRACINGS.

In describing and interpreting tracings, it is necessary to have standard points for measurement. The following points and spaces have been standardised by Mackenzie, and it is a good plan, when solving tracings, to make a practice of systematically using them.

" Diseases of the Heart," third edition, Jas. Mackenzie.

Referring to Fig. 7:

Point 1 marks the beginning of auricular contraction.

Point 2 marks the hardening of the ventricles as they begin to contract. This point is seen in the apex tracing, Fig. 11.

Point 3 marks the opening of the semilunar valves and the commencement of the carotid pulse.

Point 4 marks the beginning of the radial pulse.

Point 5 marks the closure of the semilunar valves.

Point 6 marks the opening of the tricuspid valve. This is usually a well-defined point, occurring at the summit of the ventricular or V wave.

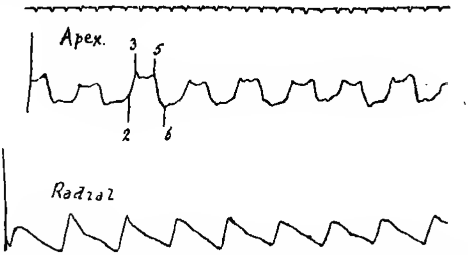

Fig. 11.—Simultaneous Tracings of Apex-Beat and Radial Pulse, showing the Standard Points of Measurement 2, 3, 5, and 6.

In all my own polygraphic tracings reproduced in this book each division of the time marker is $\frac{1}{5}$ second.

The space between points 2 and 3 is called D, or the presphygmic period of the ventricular systole.

The space between points 3 and 5 is called E; this is the period of ventricular output, called the sphygmic or pulse period.

The space between points 5 and 6 is called F; the postsphygmic period.

The space between point 6 and 2 is called G; this is the period of ventricular diastole.

In interpreting venous tracings I have found it very important always to approach a tracing in a routine and systematic manner, and have adopted the following method (still referring to Fig. 7):

1. Examine the radial tracing to see if it is regular in both its rhythm and the force of its contraction.

2. Mark in point 4, at the beginning of the upstroke in the radial tracing.

3. Mark in point 3, $\frac{1}{10}$ second before 4. Point 3, the beginning of the carotid pulse, usually occurs $\frac{1}{10}$ second before the radial. This measurement can always be checked by taking a tracing of two or three of the carotid beats.

4. With a pair of compasses measure the distance from the ordinate at the beginning of the radial tracing to point 3. With the compasses fixed, mark in point 3 on the jugular tracing, measuring from the ordinate at the beginning of the tracing. Point 3, in a normal tracing, will fall at the beginning of the carotid wave *c*.

5. From point 3 in the jugular tracing mark in point 1, $\frac{1}{5}$ second before point 3. This will normally fall at the beginning of the auricular wave *a*. The auricle normally begins to contract $\frac{1}{5}$ second before the beginning of the carotid pulse. The space between 1 and 3 is known as the A–C interval, and is taken as the measure of time that it takes the stimulus to pass from the auricle to the ventricle. I shall return to this measurement shortly.

6. Now mark in point 6 on the jugular tracing. This is usually a well-defined point. It is the point at which the V wave just begins to fall. On the radial tracing point 6 usually falls near the bottom of the aortic notch; it should always be checked by this measurement. From the beginning of *c* wave (point 3) to the summit of *v* wave (point 6) is usually $\frac{2}{5}$ second. This is a useful method of checking point 6.

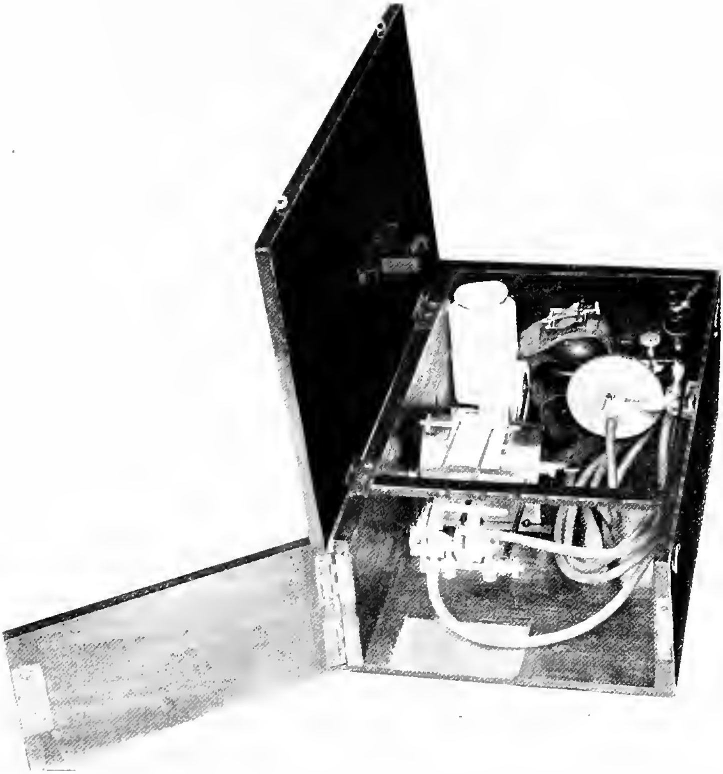

FIG. 12.—THE NEW POLYGRAPH BOX WITH INSTRUMENT IN POSITION OF DISUSE.

The instrument is kept assembled ; it is only necessary to lift it forward and fill the pens before using it.

To face p. 70

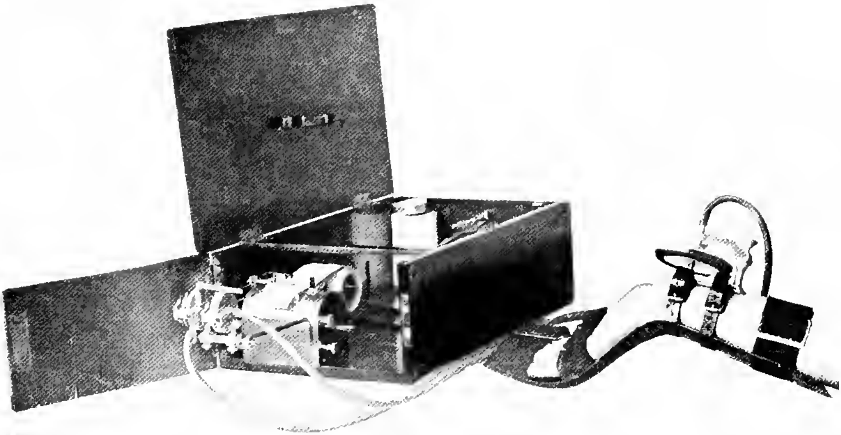

FIG. 13.—THE NEW POLYGRAPH BOX WITH INSTRUMENT IN WORKING POSITION, AND THE WRIST-SPLINT FIXED ON THE PATIENT.

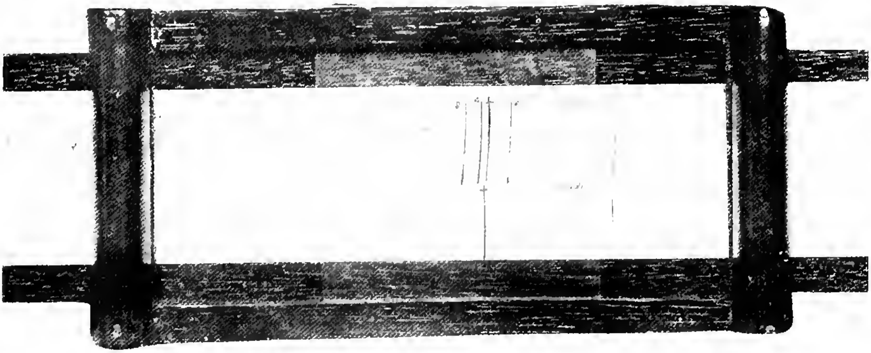

FIG. 14.—THE POLYGRAPH SLIDE-RULE WITH A NORMAL TRACING ATTACHED TO IT.

(For description of how to use slide-rule see the text.)

To follow Fig. 12

7. Point 5 is not of much practical importance in solving tracings. When present, it is represented by a notch on the V wave about $\frac{1}{10}$ second before point 6.

8. The summit of the V wave has a constant relation throughout a tracing to the beginning of the C wave. It is of some importance to bear this in mind when dealing with an irregularly contracting heart.

In the attempt to solve a difficult tracing it will frequently be found very helpful to construct a diagrammatic representation of the tracing, such as is shown in Fig. 32 and many of the other diagrams.

The down strokes in the space A represent the beginning of the *a* waves, and those in the space V the beginning of the *c* waves. The oblique lines in the space *a–c* which join the down strokes together represent the time the stimulus takes to pass from auricle to ventricle.

Lewis describes the following method of calculating the rate of the pulse from a radial tracing: with a pair of calipers measure thirty $\frac{1}{5}$ second on the time marker. Take this measured distance down to the radial tracing, placing the left point of the calipers at the beginning of the rise of a radial beat. Now count the full pulse-beats between the two points of the calipers, allowing ·1, ·2, ·3, and ·4, etc., for the fraction. If 9 whole pulse-beats and ·4 of a pulse-beat are covered between the two points of the calipers, the pulse-rate is 94. The error of this measurement is approximately only 1 in 60.

If tracings are systematically attacked in this way, the great majority will be capable of solution.

I have recently tried to simplify polygraphic technique by introducing: (*a*) a new polygraph box in which the instrument can be carried about fixed ready for use (see Figs. 12 and 13); (*b*) a wrist splint, which throws the radial artery into prominence, and also serves

Lancet, Feb. 1, 1919. 176 and 177.

to keep the radial pen from moving off the paper (see Fig. 13); (*c*) a modified slide rule for measuring out the tracing (see Fig. 14). This consists of a piece of polished wood with grooved sides to carry a slide. The slide has a window in it which is partially filled with a thin sheet of celluloid; a space is left between the celluloid sheet, and the ends of the frame of the slide, so that the waves can be marked on the tracing with a pencil. Fig. 14 shows the slide rule with a tracing in position. The tracing is fixed at each end of the slide rule by drawing-pins. The slide is then slipped on and moved over the tracing so that the celluloid sheet lies over the ordinates. The ordinates of both the radial and venous curves are then traced on to the celluloid. A line $\frac{1}{10}$ second to the left of and parallel to the ordinate of the venous tracing is marked in, C, and gives the position of the beginning of the carotid wave when the radial ordinate is placed over the beginning of the rise of any of the radial beats. Another ordinate, A, $\frac{1}{5}$ second to the left of the carotid ordinate, marks the position of the beginning of the auricular wave. An ordinate $\frac{2}{5}$ second to the right of the carotid ordinate gives approximately the summit of the V wave. Any of these points can immediately be fixed in any particular part of the tracing by moving the slide until the radial ordinate lies over the beginning of the rise of the corresponding radial pulse. Similarly any two consecutive radial, carotid, or auricular waves can be marked on the celluloid sheet and the spacing rapidly compared throughout the whole tracing by moving the slide over it. Any other of the many measurements that may be necessary can be made rapidly in the same way. The most convenient method of marking in these ordinates is to use a copying pencil, but if ink is preferred the best is Arnold's black waterproof drawing ink.

THE HIRSCHFELDER OR A. G. GIBSON WAVE.

A wave with which the reader must be familiar is the *h* wave occurring early in diastole, first described by Hirschfelder in 1907, and attributed by A. G. Gibson to the momentary closure of the tricuspid valves caused by the sudden inrush of blood from the auricle to the ventricle. This wave, when present, usually occurs after the V wave (see Fig. 15, waves marked H).

Johns Hopkins Hosp. Bull., 1907, xviii., 265-267. *Lancet*, 1907, ii., 1380-1382.

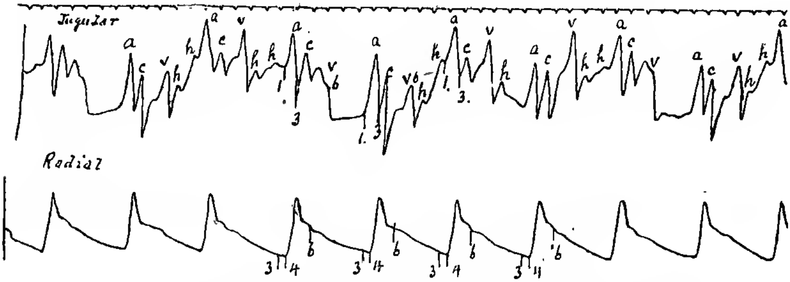

FIG. 15.—SIMULTANEOUS TRACINGS OF JUGULAR AND RADIAL PULSES. The jugular tracing shows Hirschfelder or A. G. Gibson waves *h* as well as *a*, *c* and *v* waves.

A Hirschfelder wave may cause considerable confusion in the attempt to solve a tracing, unless the following conditions, under which it may occur, are borne in mind:

1. In a slowly beating heart the H wave may fall midway between two auricular waves and give rise to the false supposition that the case is one of partial heart-block, in which the ventricle fails, every other beat, to contract in response to an auricular stimulus.

"The Schorstein Lecture," by T. Wardrop Griffith, *B.M.J.*, Oct. 12, 1912.

2. When the heart increases in rapidity, the shortening of the cardiac cycle takes place at the expense of the diastolic period, so that an A wave may fall on the top of an H wave. It is then impossible to say exactly where the A wave begins, and it may give rise to the belief

that the A–C interval is increased, unless the method of measuring this A–C interval is adopted, which I shall soon describe.

3. In cases in which the A–C interval is increased considerably, an H wave may occur as a post-auricular wave between the A and C waves. This wave is usually only small, for there is a dilating auricle interposed between the tricuspid valve and the jugular vein.

4. In cases of heart-block a well-defined H wave may occur after those auricular contractions which are not followed by ventricular contractions. This wave must not be mistaken for a ventricular wave

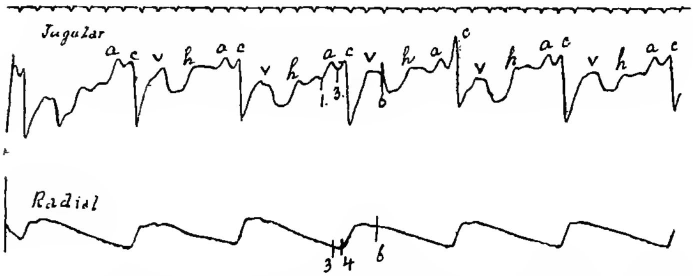

FIG. 16.—SIMULTANEOUS TRACINGS OF JUGULAR AND RADIAL PULSES, SHOWING WELL-MARKED HIRSCHFELDER OR A. G. GIBSON WAVES IN THE VENOUS TRACING, MARKED *h*.

5. In cases of auricular fibrillation in which the H wave is present, it may be mistaken for an auricular wave, and give rise to a false interpretation of the tracing. In these cases the space between the V and H waves is constant, whereas the space between the H wave and following C wave is variable, because the rhythm in cases of fibrillation is always irregular, except in some cases in which the heart is under the influence of digitalis and when fibrillation occurs with heart-block. But in these two exceptions the rhythm is slow, and the H wave would

be so far separated from the C wave that it could not be mistaken for an A wave.

6. The H wave may produce a third sound heard only at the apex.

Fig. 16 shows a well-marked Hirschfelder wave. The tracing was taken from a man suffering from typhoid fever, pulse-rate 50 per minute.

THE MEASUREMENT OF THE A–C INTERVAL.

As the measurement of the A–C interval is the way we estimate the function of conductivity of the auriculo-ventricular bundle, it is important that it should be accurate. The usual method of estimating the A–C interval is to take the distance between points 1 and 3.

Point 1, the beginning of auricular contraction, is very often ill-defined. For example, suppose the auricular wave to fall on an H wave, or on the summit of a V wave, it is thus impossible to say where the auricular wave begins; and, under these conditions, it may be assumed erroneously that the A–C interval is increased as the result of some pathological lesion in the bundle of His. Moreover, the wave C does not always represent the carotid pulse.

Dr. Wardrop Griffith has described a very much more accurate method of measuring the A–C interval, which has the great advantage of being independent both of the knowledge of the point at which the auricle begins to contract and of the origin of the wave C. He calls it "the summit of the auricular wave to the beginning of the radial rise (point 4) measurement." With a pair of compasses, take the measurement from the ordinate at the beginning of the jugular tracing to the summit of the A wave, and transfer this measurement on to the radial tracing; then the distance from this point to point 4 is

Heart, vol. iii., Feb., 1912; and "Schorstein Lect.," 1912. *B.M.J.*, Oct. 12, 1912.

the measurement of the true A–C interval. Look at Fig. 15; if the heart rate were increased, the A wave would fall on the top of one of the H waves. If, then, the distance between 1 and 3 was taken as the measurement of the A–C interval, this would appear to be considerably increased.

Since an increase in the A–C interval is frequently the earliest sign that the heart muscle is being attacked by disease, it is important to insist on the accuracy of the method of measuring this interval. Fig. 17 illustrates this point. The A–C interval in the wave I have marked measures $\frac{3}{10}$ second. As this patient was a boy of eighteen years complaining of symptoms now classified under the term "effort syndrome," from a superficial examination of this tracing one might have thought the A–C interval was lengthened, and concluded that there was active disease affecting the heart muscle. But if Griffith's "summit of A to beginning of radial rise" is taken it will be seen that the true A–C interval is normal.

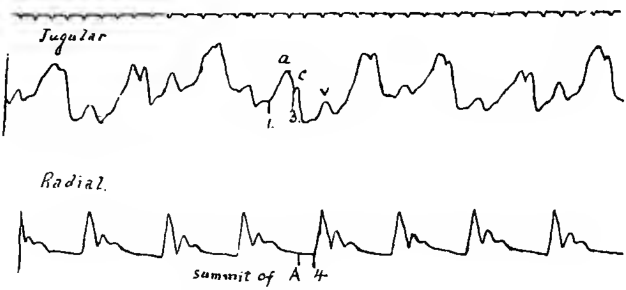

Fig. 17.—Simultaneous Tracings of Jugular and Radial Pulses.

The venous tracing shows an apparent increase of the *a-c* interval, which, however, is shown to be within the limits of the normal by applying Griffith's measurement from summit of *a* wave to the beginning of radial rise.

ELECTROPHYSIOLOGY.

Phil. Trans. Lond., 1773, 461–477.

The science of animal electricity may be said to have been founded by Galvani in 1792, when he summarised his epoch-making experiments in "De Viribus Electricitatis in Motu Musculari Modena." John Walsh had previously observed in 1773 that the *Raia torpedo*, of which there are three species inhabiting the Mediterranean and Atlantic, has the power of producing electric discharges physiologically. It has an electric organ on the back of its head consisting of laminæ composed of 800 or 1,000 polygonal cells supplied with four large bundles of nerve fibres. As early as 1678 Swammerdam showed the Grand Duke of Tuscany that the muscle in a frog's leg, when subject to an electric current, contracted. He hung a portion of muscle of the leg by a thread of nerve, bound with silver wire, and held it over a copper support, so that both nerve and wire touched the copper; the muscle immediately contracted. Galvani was not, however, aware of this experiment when he announced his discovery that, when two dissimilar metals are placed in contact with a nerve and a muscle respectively, and then brought into contact with each other, a contraction of muscle takes place. Galvani attributed this to the electricity generated in the frog's leg itself, but Volta, professor in the University of Pavia, proved that it arose from the contact of the dissimilar metals. Though Galvani in his later writings allowed that the electricity operating in the frog's leg arose from the two dissimilar metals employed, he insisted on there being an "animal electricity" inherent in the muscular and nervous structures, for he proved that contractions would be produced without using any metals, simply by touching a nerve at two different points along its length with a piece of muscle cut from a living frog;

and that contraction in a muscle can be caused by a conductor of one metal joining a nerve to a muscle. Galvani thought these experiments disproved Volta's contact theory. Volta regarded them as proving that the contact between nerve and muscle itself produced, "as in the case of two different metals," opposite electrical conditions. Volta in his "Letters on Animal Electricity" in 1792 divided conductors of electricity into metallic and liquid, invented the voltaic pile, and showed that a muscle can be thrown into tetanic contraction by successive electrical stimulations.

Nobili of Florence, in 1825, showed that when a nerve and muscle of the frog are respectively connected by a water-contact with the terminals of a delicate galvanometer a current is produced which lasts several hours.

'Electro-cardiography and phonography," by Lewellys F. Barker, *Johns Hopkins Hosp. Bull.*, 1910.

Matteucci, who introduced the word "tetanise" in 1838, proved that there may be an electromotive force through the muscle alone. Except for the researches of Nobili and Matteucci the discoveries of Galvani, after the first outburst of enthusiastic and exaggerated hopes which they aroused, suffered from neglect. It was not until the middle of the nineteenth century that the method of electrophysiology was really established, or it may be said founded, by Émil du Bois Reymond of Berlin, who held the chair of physiology from 1858 for the rest of his life. His studies were chiefly connected with the physiology of the muscle nerve preparations which he did so much to introduce into laboratory experimentation; they were twice printed in a collective form—1848–60 and 1883. One of the most interesting of his demonstrations is, that, if the end of a muscle be cut across, the cut end is electrically negative and the undamaged sides electrically positive; and that the difference of potential will produce a current even while the muscle is at rest. He used a fine astatic galvanometer (after Nobili's method) with 20,000 turns of wire in its coils. He also obtained currents from his own muscles by dipping the tips of his forefingers in two cups of salt water which he put in communication with the galvanometer terminals. If there was a sudden contraction of the muscles of either arm, it gave rise to a current running from the contracted to the uncontracted muscles.

A great interest in the subject of electrical investigation of muscle was aroused by Reymond's work, but, as in the case of Galvani's discoveries, the enthusiasm was so intense at the time and such exaggerated hopes were entertained which were not destined to be immediately fulfilled that a violent reaction set in, even before the death of du Bois Reymond himself. Perhaps in consequence of the double reaction, expectation and enthusiasm in regard to electrophysiological research was kept within sober limits, lest the progress of that branch of science should again be endangered, for Reymond certainly put it on a sufficiently firm basis. It is, however, now established that it is profitable to physiology and medical practice to make a study of the electric changes which occur in the body; and the most promising line of investigation is that of research in the electrical variations of the heart muscle.

The so-called "resting current," or the electrical changes which are presented by the human body when at rest, are of interest; but even more interesting is the "active current" produced during excitation.

In 1856 an interesting demonstration had been made by Kolliker and Müller, who showed, by means of the rheoscope, the rhythmic current variations in the beating heart of a frog, and they observed that during ventricular

Verhandl. d. phys.-med. Gesellsch. in Würzburg, Bd. vi., S. 528.

contraction there were two elevations in the same direction on the curve.

Phil. Trans. Roy. Soc., 1889, clxxx., 169-194.

A greater step was made by A. D. Waller in 1889 when he demonstrated the possibility of registering by the galvanometer the action currents emanating from the human heart. He obtained the curves by photographing the movements of the mercury meniscus of a capillary electrometer, the most sensitive apparatus available in his time. Owing, however, to the inertia of the mercury it was necessary to correct the curve so obtained by elaborate mathematical calculations in order to obtain a true reading of the electrical variations.

"Un nouveau galvanometre," *Arch Néerlandaises des sciences exactes et naturales*, ser. ii., tome vi., p. 625, 1901.

This method was perfected in 1901 by Wilhelm Einthoven of Leyden by his invention of an extremely sensitive instrument, "the string galvanometer," on which the modern electrocardiograph is founded.

In studying the Lippmann capillary electrometer he (Einthoven) perceived that it had not the necessary quickness of response to a given strength of potential when sensitive enough for very feeble currents; or if sufficiently responsive it became insufficiently sensitive. He set to work, therefore, to find an instrument which, besides being entirely aperiodic, must be practically free from lag or inertia, and be capable of responding to the most minute electric currents. Following the suggestion of Ader concerning an instrument used in international telegraphy, he reversed the former condition in galvanometers in which the magnet is movable and the measurable current passes through stationary coils, and substituted a stationary electro-magnet, whilst the current to be measured passes through a movable string.

His instrument consists essentially of a very delicate thread of platinum or silvered glass which is stretched from supports, and hangs in a narrow gap midway between the poles of a stationary electro-magnet. When the feeblest heart currents pass through this delicate thread they cause deflections which are smaller or greater in proportion to its state of tension. The records of cardiac excitation are called "electrocardiograms," and they may be compared to an accurate telegram—*i.e.*, bulletin—of the heart's electromotive condition.

This instrument, as well as being "dead beat," is so extremely sensitive that it will detect a current less than one sixty thousand millionth of an ampère.

Lancet, May, 1909.

It is interesting to know that Waller, writing in the *Lancet* of May, 1909, after a vist to Leyden to see Einthoven's work, seems not to have foreseen any clinical application of electrocardiography. He writes: "I do not, however, imagine, nor, I believe, does Einthoven, that the string galvanometer, however useful and nccessary it may be in the physiological laboratory, is likely to find any extensive use in the hospital as affording means of physical diagnosis more searching than those in daily use. It can at most be of rare and occasional use to afford a concrete graphic record of some rare anomaly of cardiac action, as it has already in the hands of Einthoven, Lewis, and others, served to bring into clear evidence the extraordinary disorders of contraction of which the diseased heart is capable. But for all ordinary purposes of diagnosis the finger-tips of the physician will hardly be helped by an instrument as difficult to manage and to interpret as is the string galvanometer."

Before considering the electrical variations of the heart it will be convenient to inquire into the deflections of the galvanometer caused by the passage of a wave of excitation through a strip of muscle.

If a strip of muscle is connected to a string galvanometer by two non-polarisable electrodes, and one end stimulated, the wave of excitation which passes along the muscle will cause a deflection of the galvanometer string as soon as it reaches the first electrode. The deflection is in the same direction as would occur if the excited part of the muscle was replaced by a zinc terminal and the quiescent part by a copper terminal in a zinc-copper cell. We therefore say that excited or active muscle is electrically negative to quiescent muscle. When the excitation wave passing along the strip of muscle reaches the second electrode, the potential is equal at the two electrodes, so the galvanometer string returns to zero; the excitation wave next leaves the first but remains at the second electrode, when

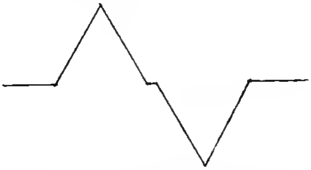

FIG. 18.—DIAGRAM OF THE DIPHASIC CURVE PRODUCED BY AN EXCITATORY WAVE WHICH STARTS AT ONE END OF, AND PASSES ALONG, A STRIP OF MUSCLE WHICH IS LED OFF AT EACH END BY TWO NON-POLARISABLE ELECTRODES TO A STRING GALVANOMETER.

again a difference of potential occurs, but this time the muscle under the second electrode is relatively negative to that under the first, so that the galvanometer string moves in the opposite direction; the wave of excitation now leaves the second electrode and the galvanometer again moves back to zero. Thus an excitation wave passing through a strip of muscle which is led off by two non-polarisable electrodes to a string galvanometer gives a diphasic curve represented by Fig. 18.

The direction in which the string of the galvanometer moves depends therefore upon three factors: (*a*) the point at which the excitation wave starts, (*b*) the direction it takes, and (*c*) the relative position of the two electrodes leading off to the galvanometer.

The " extent " to which the string moves depends upon the difference of potential between the two electrodes, and this is proportional to the space of time which elapses while the "excitation wave" passes from one electrode to the other; the longer the time the greater the opportunity for the difference of potential to develop in the galvanometer. Therefore, as we shall see later, the extent of the deflection cannot be any indication of the force of contraction.

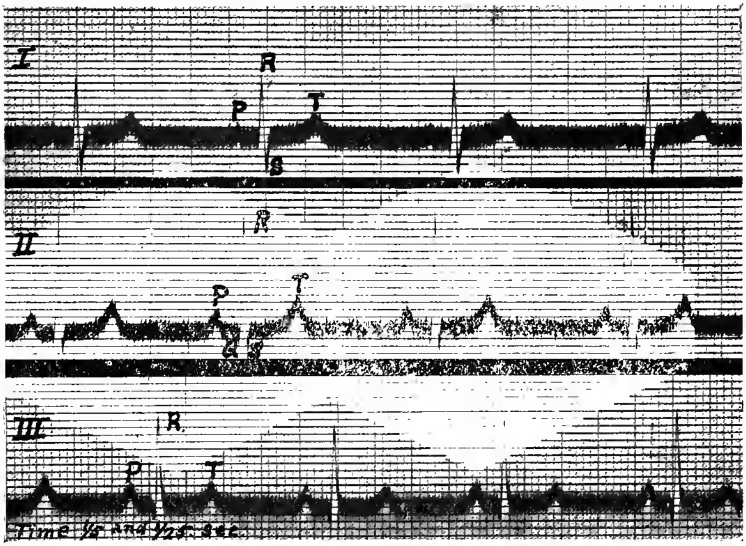

FIG. 19.—A NORMAL ELECTROCARDIOGRAM, SHOWING LEADS I, II, AND III, AND THE DEFLECTIONS P, Q, R, S AND T.

The normal electrocardiogram, of which Fig. 19 is an illustration, is divided into two parts:

1. The "auricular complex," which extends from the

commencement of the rise P to the beginning of Q, or, if Q is absent, the beginning of R.

2. The "ventricular complex," which extends from the beginning of Q (if present) or R to the end of the wave T, and consists of the deflections Q R S T.

The letters P Q R S T were originally introduced by Einthoven and should at present be retained, as they do not attempt to indicate the cause of the deflections in the electrocardiogram.

Heart, 1909–10, i., 235-261. Gotch, as we shall see presently, thought the normal excitation wave in the ventricle started at the venous part of the base, passed down to the apex, and then back again to the base at the origin of the aorta. We shall see later, when considering the work of Lewis on conduction of the excitation wave in the ventricle, that Gotch's view is incorrect, that the excitation wave spreads almost simultaneously over the whole subendocardial surface of the ventricular musculature, and that the time of its appearance at different points on the superficial surface is dependent upon the varying thickness of the ventricular muscle.

Seemann, after carrying out a number of experiments on the frog in 1912, concluded that the initial deflection R in the ventricular complex is the electrical expression of the onset of excitation; but he regarded the final deflection T as the electrical expression of contraction. Mines held that Seemann's hypothesis was completely disproved by the experimental demonstration that muscular contraction of the ventricle can be completely abolished, whilst the final electrical wave T remains normal. He (Mines) believed that the cause of the final deflection wave T was that the offset of the excitation process did not take place simultaneously over the ventricle.

Journ. Physiol., xlvi., 1913, pp. 188-235.

Burdon-Sanderson and Page, in their experiments on the frog and tortoise in 1880 and 1883, showed that by locally warming the muscle under one electrode, the curve given by the capillary electrometer after the muscle was stimulated was changed as it would be if there were a quickening in the passing off of the excitation process under the warmed electrode. It was this experimental result which led up to our present belief of the cause of the wave T, as expressed above by Mines.

Journ. Physiol., vol. iv., p. 327.

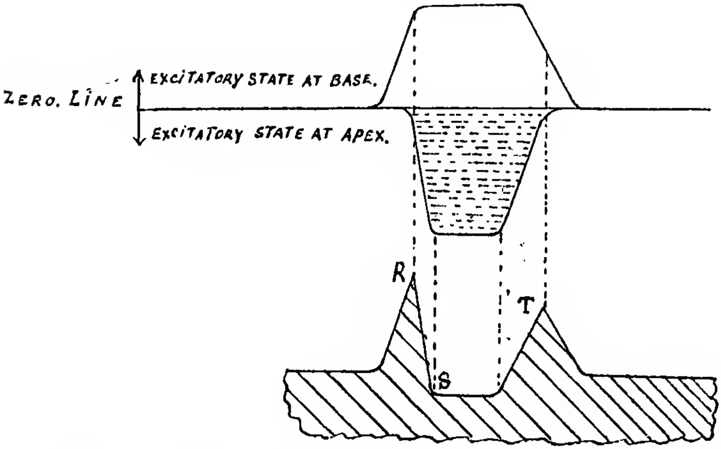

FIG. 20.—DIAGRAM TO SHOW MANNER OF PRODUCTION OF NORMAL ELECTROCARDIOGRAM BY LONG DURATION, WITH LESS HEIGHT OF EXCITATORY STATE AT THE BASE THAN AT THE APEX.

Modified from "Principles of General Physiology," by W. M. Bayliss. London: Longmans.

Bayliss and Starling in 1892 concluded that the form of the final deflection of the ventricular complex is determined by the unequal duration of the excitation process at the base and apex of the ventricle; and that the excitation process normally lasts longer at the base than at the apex.

At present it is only safe to say that the T wave is caused by the termination or passing away of the excitatory process. An unsuccessful attempt was recently made to explain all the deflections of the electrocardiogram by regarding the negativity at the base as lasting longer, but being of less height than that at the apex. Fig. 20 is a diagrammatic representation of this theory.

"The Spread of the Excitatory Process in the Vertebrate Heart," parts i.-v., by Thos. Lewis, *Philosophical Transactions of the Royal Society of London,* series B, vol. ccvii., pp. 221-310.

Lewis has succeeded in showing which ventricle is responsible for the production of the Q R S deflections in the three standard leads. The excitatory process reaches the right and left ventricles by the right and left branches respectively of the *a–v* bundle, and spreads all over the inner surface of the heart by the subendocardial network known as the Purkinje system. The stimulus travels along the Purkinje system five times more rapidly than it does through the cardiac musculature. On the right side the excitatory process starts at the upper part of the septum and spreads over the inner surface of the right ventricle in a clockwise direction; on the left side it passes in an anticlockwise direction. The passage of the excitatory process over the two ventricles gives rise to two electrical forces, different in direction. The direction of the resultant of these two electrical forces, or differences of potential, determines the deflections of the electrocardiogram.

When the main right or left branch of the *a–v* bundle is cut or damaged to such an extent that it will not conduct a stimulus, it is possible to obtain a record of the Q R S deflections of the sound ventricle only. These electrocardiograms of only left or right ventricle are known as levograms or dextrograms respectively. And as the normal electrocardiogram, or bigram, is the algebraic summation of the levogram and dextrogram, by a study of the levogram and dextrogram in the three leads we are able to allot the share which each ventricle plays in producing the Q R S deflections in the bigram.

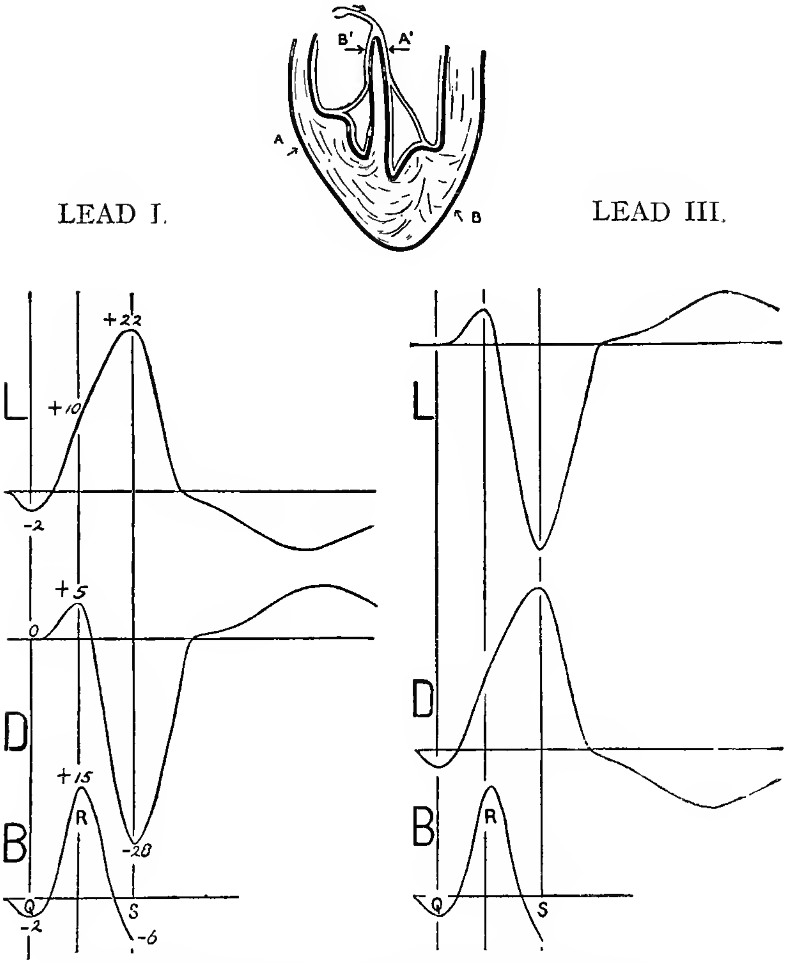

Fig. 21.—A Diagram of the Ventricles seen in Section and of the Auriculo-Ventricular Bundle and its Branches.

A lesion of the bundle division at A′ forces the natural impulse to descend solely through the right division, the supply of the right ventricle. The corresponding electrocardiograms are characteristic, and are not dissimilar in form to those obtained by stimulating the right ventricle at A. A lesion of the right bundle division at B confines the descending impulse to the path of the left division. The

The levograms and dextrograms hold true only for the rapid Q R S group of deflections, and not for the deflection T. This is because the T wave occurs late in systole, so that the excitatory process has time to spread from the sound side of the heart to the damaged side by way of the muscle fibres.

Fig. 21 and the deductions are taken from "Clinical Electrocardiography," by T. Lewis.

In lead I.:

Q is a left ventricular event.
R is mainly a left ventricular event.
S is a right ventricular event.

In lead III.:

Q is a right ventricular event.
R is mainly a right ventricular event.
S is a left ventricular event.

A SUMMARY OF THE VARIATIONS WHICH MAY OCCUR IN NORMAL ELECTROCARDIOGRAMS.

The standard leads adopted in electrocardiography are as follows:

Lead I.: Right arm and left arm.
Lead II.: Right arm and left leg.
Lead III.: Left arm and left leg.

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corresponding curves are characteristic and are not dissimilar in form to those obtained by stimulating the left ventricle at B.

Below the diagram are two series of curves, arranged vertically and corresponding to leads I. and III. The top curve in each series is the *levogram,* obtained when the right bundle division is damaged; the middle curve in each series is the *dextrogram,* obtained when the left bundle division is damaged. The bottom curves represent the initial phases of the normal electrocardiogram and the diagram is arranged to show that these initial phases are the algebraic product of the levogram and dextrogram. In the series from lead I., I have inserted the amplitudes of the curves where they cross the common vertical lines, marking them as + or – quantities according as the deflection is up or down. Thus Q (– 2) is the result of a similar dip (– 2) in the levogram; R (15) is the product of the small spike (5) in the dextrogram and of the rising levogram (10).

By permission from "Clinical Electrocardiography," by T. Lewis London: Shaw and Sons.

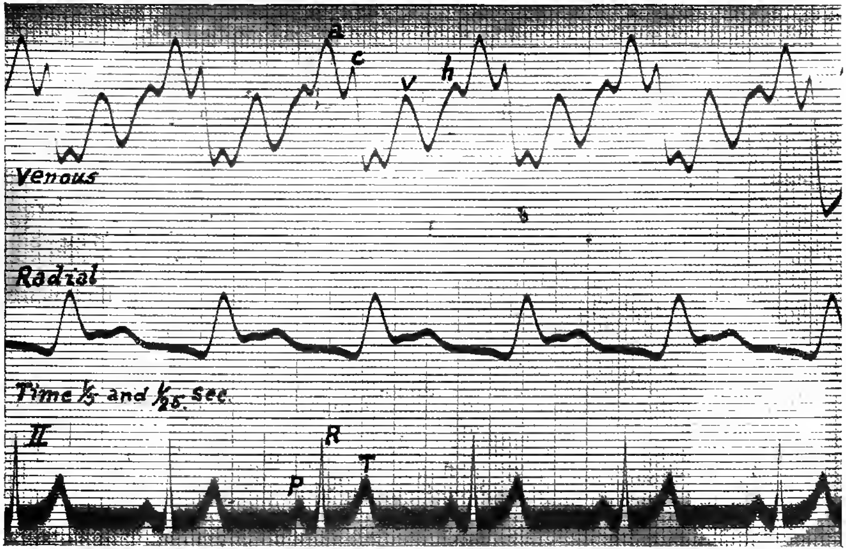


FIG. 22.—COMBINED VENOUS, ARTERIAL, AND ELECTROCARDIOGRAPHIC TRACINGS FROM A NORMAL SUBJECT.

Fig. 19 shows electrocardiograms from the three leads in a normal subject; and Fig. 22, also taken from a normal subject shows, combined venous, arterial, and electrocardiographic tracings.

"Clinical Electrocardiography," by T. Lewis.

The following is a brief summary of the variations which may occur in the deflections of normal electrocardiograms:

The deflections P and R are directed upwards in the curve if the connections to the patient are made in such a way that, if the right arm is replaced by a zinc terminal, and the left leg by a copper terminal, an upward deflection will occur.

The deflection P is small, and in normal hearts rarely exceeds two or three scale divisions.

P in normal subjects is always upright in leads I. and II., but not always in lead III., so that an inverted P in lead III. does not necessarily imply an ectopic contraction.

Deflections R and T are always present, but Q and S are not always present.

R is always directed upwards, and is normally most prominent in lead II.

S is directed downwards.

In healthy individuals T is always upright in lead II., and usually so in lead I., but is often inverted in lead III., where it is usually associated with a bizarre Q R S group. Inversion of T is always pathological in lead II., and sometimes also in lead I. The inversion in lead II. is usually of ill-omen, and patients presenting it have been short-lived.

Normal electrocardiograms occasionally exhibit notching of R and S, and in lead III. bizarre types of the initial deflections Q R S are not uncommon. The Q R S group of deflections does not exceed $\frac{1}{10}$ second in normal subjects.

The P–R interval varies from ·12 to ·18 second; it never exceeds ·2 second in health.

A deflection U is not uncommon, and may be prominent; it is related to the early events in diastole.

All the deflections vary in amplitude and shape. Considerable changes in the size or direction of deflections in electrocardiograms taken under similar circumstances do not occur in either healthy or diseased hearts. An observed change is significant of altered function. If electrocardiograms which show considerable divergence from what is regarded as normal are obtained, it is probable that the heart is abnormal. Hearts which are gravely affected rarely yield normal electrocardiograms.

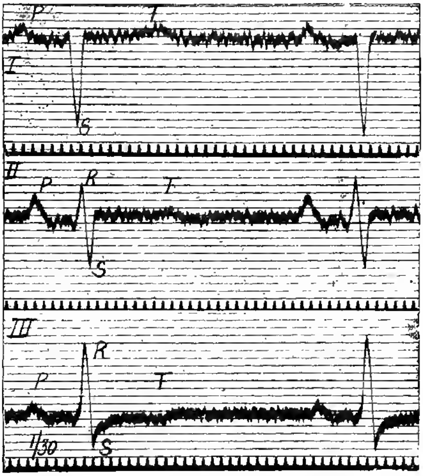


FIG. 23.—CURVES FROM THE THREE LEADS IN A CASE WHERE THERE WAS HYPERTROPHY OF THE RIGHT VENTRICLE.

Note that there is but a trace of R in lead I. and that S is very deep; also that R is tallest in lead III., while S is small.

(From "Clinical Electrocardiography." T. Lewis.)

Advancing age modifies electrocardiograms chiefly by a decrease in the amplitude of T.

Exercise modifies the electrocardiogram by increasing the amplitude of T.

By a study of the levograms and dextrograms in Fig. 21

it is possible to deduce the changes which will occur in the electrocardiogram resulting from certain alterations in the cardiac mechanism—*e.g.*:

The modifications of the curves which occur in relative hypertrophy of one or other ventricle are—

1. Relative hypertrophy of right ventricle (see Fig. 23):

The tallest deflection, R, is in lead III.
The deepest deflection, S, is in lead I.
The deflection Q is most prominent in lead III.

2. Relative hypertrophy of left ventricle (see Figs. 24 and 25):

The tallest deflection, R, is in lead I.
The deepest deflection, S, is in lead III.
The deflection Q is most prominent in lead I.

In relative hypertrophy of either right or left ventricle the time of the initial ventricular deflections Q R S is normal, not exceeding $\frac{1}{10}$ second.

The modifications which occur in a lesion of the right or left branch of the *a–v* bundle are—

1. Lesion of the right branch of the *a–v* bundle (see Figs. 26 and 27):

(*a*) The initial or Q R S deflections are the same as in left-sided preponderance in each lead.

(*b*) The deflection-time of the Q R S group is greater than $\frac{1}{10}$ second and is usually $\frac{1}{3}$ or more of the total ventricular complex.

(*c*) There are two main deflections, the first representing R or S, the second T. The second deflection is in the opposite direction to the first in each lead. Notching of the initial deflection in leads I. and III. is common.

It is of some interest to compare Figs. 27 and 28. They were both taken from the same patient, who was suffering from subacute infective endocarditis of the aortic valve. Fig. 28 was taken on December 6, 1918; it

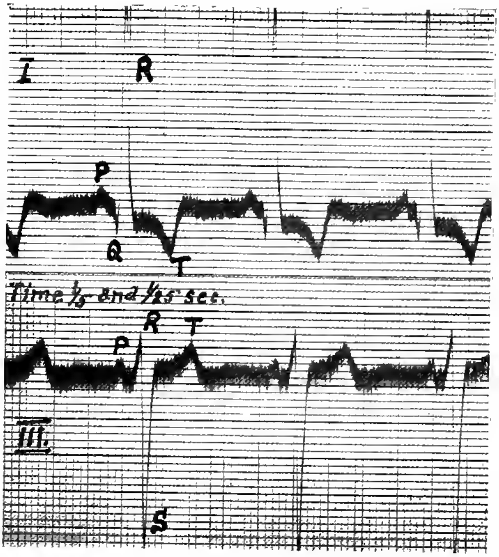


FIG. 24.—CURVES FROM LEADS I. AND III., SHOWING RELATIVE HYPERTROPHY OF THE LEFT VENTRICLE.

R is tallest in Lead I., while S is deepest in Lead III. Q is present in Lead I. only.

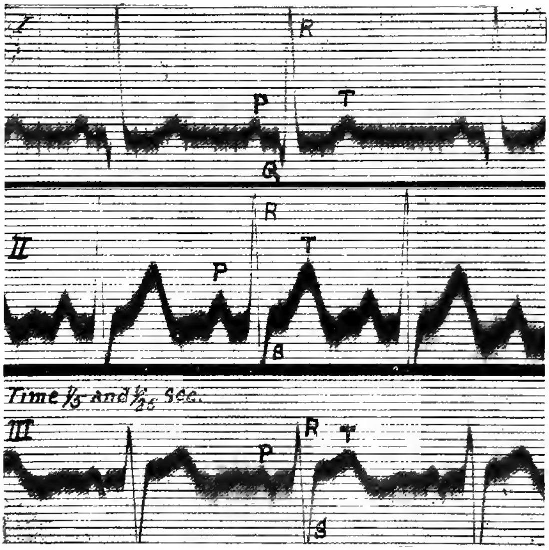


FIG. 25.—CURVES FROM THE THREE LEADS, SHOWING THE SAME FEATURES AS FIG. 24, BUT A LESS DEGREE OF LEFT VENTRICULAR PREPONDERANCE.

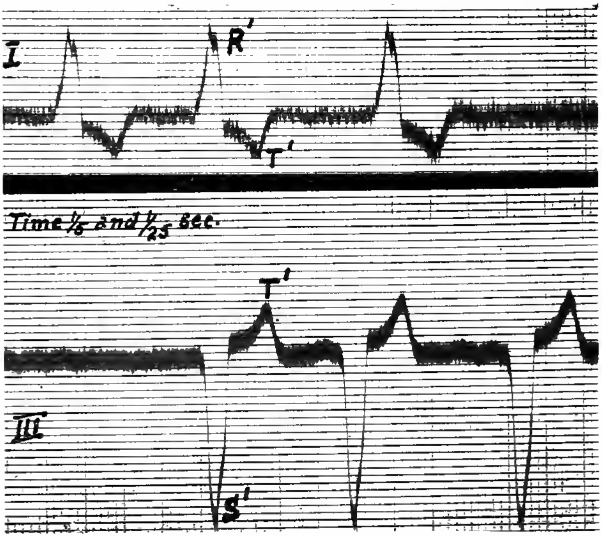


FIG. 26.—CURVES FROM LEADS I. AND III., SHOWING A DEFECTIVE CONDUCTION OF THE RIGHT BRANCH OF THE AURICULO-VENTRICULAR BUNDLE.

NOTE.—The time of the initial deflection exceeds $\frac{1}{10}$ second and the deflections R′ and S′ are large and in the opposite direction to T′. Incidentally the curves show auricular fibrillation.

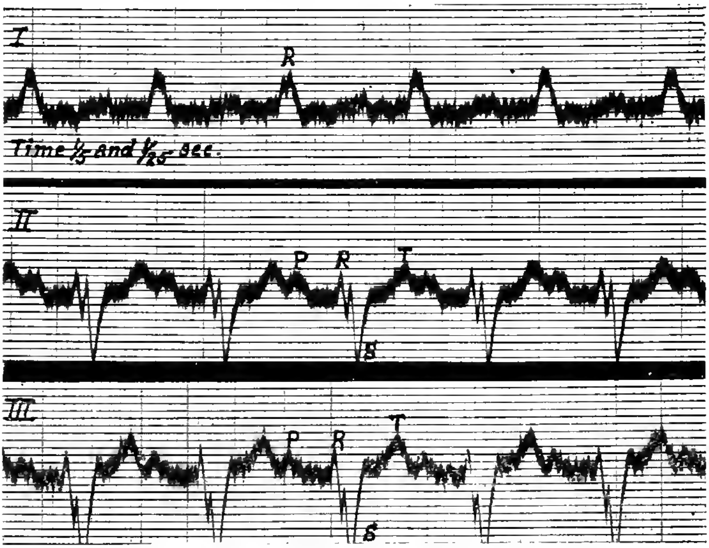


FIG. 27.—CURVES FROM THE THREE LEADS IN A CASE OF DEFECTIVE CONDUCTION OF THE RIGHT BRANCH OF THE AURICULO-VENTRICULAR BUNDLE, SHOWING THE SAME FEATURES, BUT LESS MARKED, AS FIG. 26, AND IN ADDITION A P.-R. INTERVAL OF A LITTLE MORE THAN $\frac{1}{5}$ SECOND.

does not show any sign of defective conduction of the right branch of the *a–v* bundle, for the deflection-time of the Q R S group is less than $\frac{1}{10}$ second. But there is some delay in the conduction of the stimulus in the bundle before it divides, for the P–R interval is a shade over $\frac{1}{5}$ second. Fig. 27 was taken only twenty-four days later. In leads II. and III. the deflection-time of the Q R S group is well over $\frac{1}{10}$ second.

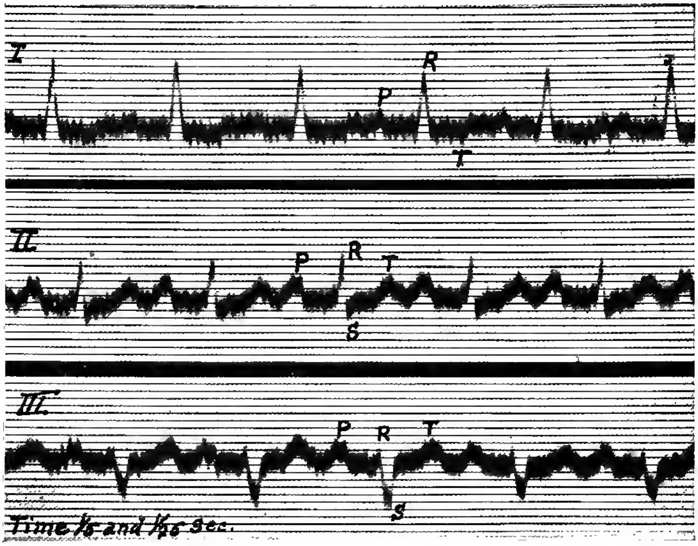


FIG. 28.—ELECTROCARDIOGRAM FROM THE SAME SUBJECT AS FIG. 27 BUT TAKEN TWENTY-FOUR DAYS EARLIER.

The curve does not show any defect in conduction of the right branch of the *a–v* bundle, but the P–R interval is a shade over $\frac{1}{5}$ second.

2. Lesion of the left branch of the *a–v* bundle (see Fig. 29).

(*a*) The initial or Q R S deflections are the same as in right-sided preponderance in each lead.

(*b*) The deflection-time of the Q R S group is greater than $\frac{1}{10}$ second, and is usually $\frac{1}{3}$ or more of the total ventricular complex.

(*c*) There are two main deflections, the first representing R or S, the second T. The second deflection is in the opposite direction to the first in each lead.

Left bundle branch lesions are very uncommon; only a few cases have been reported.

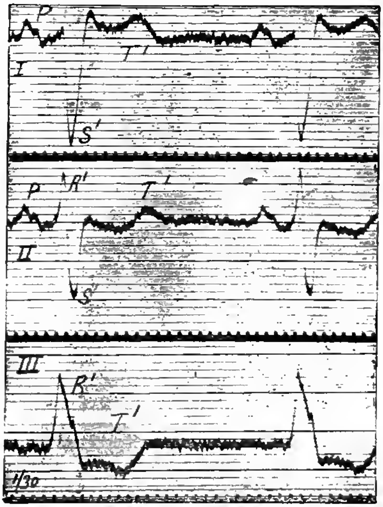


Fig. 29.—Curves from the Three Leads, evidencing Functional Defect of the Left Division of the Auriculo-Ventricular Bundle.

The initial deflection has a duration of more than $\frac{1}{10}$ second. R is tallest in lead III., S is deepest in lead I. The direction of the deflections in leads I. and III. is the reverse of those in Fig. 26.

(From "Clinical Electrocardiography." T. Lewis.)

The electrocardiogram reveals the same features in the heart-beat, both normal and abnormal, as the clinical polygraph, except that it cannot be used as a means of diagnosing pulsus alternans.

As the records of the electrocardiograph depend only upon the passage of the excitation wave through the heart, and not, as in the clinical polygraph, upon the pressure changes in the internal jugular veins, which varies from time to time and may be obscured by obesity of the neck, etc., it follows that these records are much more certain and accurate than those of the clinical polygraph. But the latter instrument gains over the former in being easily carried from house to house.

Abnormal cardiac contractions will give abnormal electrocardiographic curves, because, as we shall see later, the excitatory stimulus then arises from some point in the cardiac musculature other than the sino-auricular node. By artificially stimulating the heart muscle at certain points other than the S–A node, electrocardiograms have been produced exactly corresponding to those given by pathological abnormal contractions.

The auricular complex is normal in outline only, when the stimulus which excites contraction of the auricle arises in the S–A node.

The ventricular complex is normal in outline only when the stimulus which excites contraction of the ventricle is supraventricular—that is to say, when it arrives at the ventricle by way of the A–V bundle and its branches.

THE FIVE FUNCTIONS OF HEART MUSCLE.

Further advances in connection with the heart-beat will be most conveniently reviewed by considering separately the following five functions of heart muscle to which attention has been drawn by Gaskell:

"The Contraction of Cardiac Muscle," Schafer's "Physiol.," vol. ii.

1. The function of stimulus production, or the power to produce a stimulus.

2. The function of excitability, or the power to receive a stimulus.

3. The function of conductivity, or the power to convey a stimulus through the muscle substance.

4. The function of contractility, or the power to contract in response to a stimulus.

5. The function of tonicity, or the power to retain a certain amount of contraction even when active movement has ceased.

I take this course because I believe in doing so we shall obtain a clearer insight into the heart-beat, and also because I believe that in future it will be necessary for physicians to concentrate their attention on finding out the effect of disease on these functions, rather than to rest content with nomenclature of valve lesions, as has been too frequently the custom in the past. It is only by fully appreciating the functions of the "all-important cardiac muscle" that the physician can be guided to an intelligent prognosis and method of treatment.

1. THE FUNCTION OF STIMULUS PRODUCTION.

Since the time of Borelli discussion has never ceased as to whether the origin of the cardiac stimulus to contraction be neurogenic or myogenic. Borelli first expounded the neurogenic theory. In the time of Haller the blood flowing through the heart was believed to be the stimulus which caused the heart to beat; but when it was pointed out that a frog's heart outside the body continued to contract without any blood passing through it, physiologists then rejoined that the power of rhythmical contraction existed within the heart muscle itself. Then came the discovery by Remak, in 1848, of ganglion cells in the sinus, and in 1852 Bidder and Rosenberger described ganglion cells at the junction of auricle and ventricle; and Carl Ludwig discovered similar cells in the interauricular septum. It was natural that the neurogenic school should ascribe the cardiac contraction to rhythmical stimuli sent out from Remak's ganglion cells. Following this, Eckhard, Foster, and Dew Smith showed that the apex of the frog's heart, which contains no ganglion cells, can be made to beat rhythmically when stimulated—strong evidence in favour of the myogenic theory. In further support of this theory it was shown that the ganglion cells can be excised without interfering with the heart-beat.

Arch. f. Physiol. u. Wissensch. Med., 1848, S. 139.
Ibid., 1852, S. 172.
Ibid., 1848, S. 139.

Beitr. z. Anat. u. Physiol., Giessen, 1858, Bd. i.
Journ. Anat. and Physiol., 1876, vol. x.

The neurogenic school then opposed the upholders of the myogenic theory by taking up the attitude that the stimulus originated in the fine nerve fibres which wind round the muscle cells. Gaskell and Engelmann have been to the fore in establishing the myogenic theory; and against the theory of the stimulus originating in nerve fibres they have brought evidence that in the chick the embryonic heart beats rhythmically before the nerve cells or nerve fibres have developed in it. The experiment performed by Burrows in 1912 seems to establish the myogenic theory beyond any doubt. He showed that bits of heart muscle from embryo chicks when kept in blood plasma continued to beat for thirty days, that cells

"Croonian Lecture," *Phil. Trans.*, 1882, pp. 993-1033.
Pflüger's Arch., 1896, Bd. lxv., S. 109.

Münch. med. Woch., 1912, vol. lix., pp. 1473-1475.

wandered off from the mass and multiplied, and the newly formed cells then began to beat.

Engelmann believes that this stimulus-producing material is continually being formed in the muscle cells. The molecular explosion which occurs at systole destroys the stimulus-producing materials, not only at the point at which contraction originated, but throughout the whole heart. The molecular structure has then to be built up again to the necessary point at which contraction can occur again.

"Textbook of Physiology," ed. by E. A. Schafer, London, 1900, vol. ii, p. 197.

What, then, is the function of the ganglion cells? Gaskell answers this question in the following words: "The ganglion cells in the heart are part of the great group of ganglion cells which are situated on the course of the small-fibred efferent nerves supplying the viscera. These cells form the outlying vagrant group of nerve cells which are known by the name of the sympathetic and cerebrospinal ganglia. In the case of the heart, the ganglion cells are the cells belonging to the small-fibred efferent cardiac fibres of the vagus, just as some of the cells in the ganglion stellatum and in the inferior cervical ganglion are the cells belonging to the small-fibred efferent cardiac fibres of the augmentor nerve. There is no more need to assign special functions to these cells than to any of the other peripheral efferent nerve cells. They are cells connected only with the inhibitory fibres of the vagus, and as such are simply part and parcel of the mechanism of inhibition, just as the corresponding cells in the ganglion stellatum are simply part and parcel of the augmentor mechanism."

It is convenient here to complete the account of the nerve-supply of the heart.

In 1845, at the meeting of the Italian Scientific Investigators at Naples, the Weber brothers announced their discovery of the inhibitory action of the vagus, and described how, by stimulating the peripheral ends of the vagus nerves, they had caused the heart to stop beating. This announcement made a profound impression, as it was the first discovery of an inhibitory nerve. Schiff opposed the hypothesis of inhibition in 1849; for in his belief the vagus was a "motor" nerve, and he explained the phenomena as being due to fatigue of the heart muscle from over-stimulation of the vagus. The Webers believed that the heart was supplied by two different kinds of nerve, stimulation of which produced opposite effects, both in rate of rhythm and force of contraction, but they were unable to isolate the accelerator fibres. In 1863 Von Bezold, by dividing the spinal cord in the upper thoracic region and stimulating the cervical cord, showed that the accelerator fibres in mammals leave the cord in the region of the first thoracic ganglion of the sympathetic system. Cyon showed that this acceleratory effect was abolished by destruction of the ganglion stellatum. In 1870 Schmiedeberg and Ludwig found that stimulation in the frog of the trunk of the vagus nerve after application of nicotine to the heart produced an acceleration, and they concluded that the vagus contained accelerator fibres, but it was not until the following year that they traced out the course of the accelerator fibres in the dog, and were able to prove that the effect on the heart of sympathetic nerves was opposite to that of the vagus nerves.

Omodei's *Ann. univ. de. med.*, Milan, 1845 3, S. 227; and Wagner's "Handwörterbuch der Physiol.," 1846, iii., 45 51.

Arch. f. Physiol. Heilkunde, 1849, 8, S. 209.

Unters. über die Innervation des Herzens, 1863, 2, S. 191; and *Unters. aus d. physiol. Laborat. in Würzburg*, 1867, 2, S. 235.

Arch. f. Anat. u. Physiol., 1867, S. 389.

Ber. d. sachs. Gesellsch. d. Wiss., math. physc., ci., 1870, S. 135.

Gaskell showed that stimulation of the roots of the vagus as it leaves the medulla gives a pure inhibitory effect, and that stimulation of the sympathetic fibres between the anterior roots of the third nerve and the vagus ganglion gives pure augmentor and accelerator effects. He thus proved that the accelerator fibres join the vagus in its

Journ. Physiol., 1883, vol. iv.

course, and thus explained the results obtained by Schmiedeberg and Ludwig after applying nicotine to the heart.

Journ. Physiol., 1878, vol. i.

By modifying Roy's tonometer (which Roy first described in 1878) Gaskell was able to show that vagus stimulation diminishes the tonicity of the heart muscle; he also showed that all the other functions of the heart muscle were depressed when the vagus was stimulated. By the use of the galvanometer he was able to demonstrate that the inhibitory action of the vagus produced an electro-positive reaction, as opposed to the electro-negative reaction of stimulation of a motor nerve.

Journ. Physiol., 1887, vol. viii., p. 404.

Gaskell sums up the effect of vagal action in the following words: "Such, then, are the primary effects of stimulation of the vagus nerves: depression of rate, depression of excitability, depression of contraction force, depression of conductivity, depression of tonicity, and increased positivity of the muscular substance."

Schafer's "Physiol.," vol. ii., p. 213.

Later workers were unable to verify the increased positivity of the muscular substance of the auricle on stimulation of the vagus. Meek and Eyster in 1912 corroborated Gaskell's results, and explained the failure of other workers on the ground that in their galvanometer they did not use a string as sensitive as that of the D'Arsonval galvanometer with which Gaskell worked. They further showed that this increased positivity is not so marked on stimulation of the left vagus as on that of the right.

Meek and Eyster, *Amer. Journ. Physiol.*, 1912, vol. xxx., p. 271-277.

Although Gaskell only applied the statement just quoted to the effect of vagus stimulation upon the auricles, McWilliam, in 1888, claimed to have proved that it also applies to the ventricles of mammals, but not to all cold-blooded animals, although the nerve-supply of the heart is the same in both orders. Starling, as late as 1915, says it is still very doubtful if the vagus has a direct action on

Journ. Physiol., 1888, vol. ix., p. 345.

the mammalian ventricle, and that at any rate its effect is very slight as compared with that on the venous end of the heart. In 1913 Cohn and Lewis showed in vertebrates that, while stimulation of the right vagus slows the heart more than does stimulation of the left, yet the latter has a greater effect in depressing the conduction of the stimulus in its passage from auricle to ventricle through the *a–v* node or bundle. This result is of considerable interest developmentally, since the stimulus upon which contraction depends arises in the sino-auricular node at the mouth of the superior vena cava, whilst the auriculo-ventricular node from which the *a–v* bundle takes origin lies near the opening of the coronary sinus; these two veins represent respectively in the embryo the right and left ducts of Cuvier. Upon the recognition that stimulation of the vagus depresses the function of conductivity, especially that of the *a–v* bundle, depends the treatment of cases of simple paroxysmal tachycardia, auricular flutter and fibrillation, by digitalis. This drug, by stimulating the vagus nerves, depresses the conductivity of the *a–v* bundle, and so, by reducing the number of stimuli which are allowed to pass to the ventricle, relieves the ventricle of unnecessary and useless work. It should be noted that this slowing of the ventricle cannot be effected through the action of the vagus on the sino-auricular node, because the stimulus which gives rise to the abnormal rhythm is ectopic, and the S–A node is no longer the pace-maker of the heart.

Journn. Exper. Med., 1913, xviii., 739.

The effect of stimulation of the cardiac branches of the sympathetic (*i.e.*, the augmentor and accelerator fibres) is exactly opposite to that of stimulation of the vagus. It is interesting to notice that if the *a–v* bundle is so clamped that the ventricle responds only to every second auricular contraction, and if the accelerator fibres are then covered in mammals generally, including man, the small band of muscle fibres passing from auricle to ventricle which is now called the *a–v* bundle of His.

Journ. Anat. and Physiol., 1907, vol. xli.

In 1907 Arthur Keith and Flack described a small collection of pale, faintly striated fibres in the wall of the right auricle in the angle between the opening of the superior vena cava and the auricular appendix. This is now called the sino-auricular, or S–A, node of Keith and Flack.

"Das Reizleitungssystem des Saugethierherzens," by Gustav Fischer, Jena, 1906.

In 1906 S. Tawara described a node of tissue, similar in structure to that of Keith and Flack, lying in the wall of the right auricle between the fossa ovalis and the opening of the coronary sinus. This is now called the auriculo-ventricular, or *a–v* node, or the Knoten. He also described in greater detail the *a–v* bundle, first discovered by His, and traced its course from its origin near the *a–v* node downwards under cover of the septal cusp of the tricuspid valve where it lies in a groove on the inferior border of the central fibrous mass; it then runs forward in the pars membranacea septi along the upper border of the interventricular septum. Here it takes advantage of its position, and as it were by putting a leg down on either side of the septum, divides into right and left branches, which are finally distributed to the ventricular musculature through the subendocardial network known as the Purkinje system. The right branch always sends fibres to the moderator band, which are distributed to the anterior wall of the right ventricle. Professor J. Kay Jamieson of Leeds has kindly shown me some beautiful specimens of the distribution of the *a–v* bundle in the calf's heart. He has succeeded in injecting the fibrous sheath which surrounds the bundle and its branches, and by this means has demonstrated the minute terminal twigs distributed over the whole of the subendocardial surface.

The *s–a* node of Keith and Flack, the *a–v* node of Tawara, and the *a–v* bundle of His, are remains of the embryonic primitive cardiac tube. During development the primitive cardiac tube is twisted upon itself, and from the dorsal aspect of the auricular segment there is a bulging from which the auricles develop; on the ventral aspect of the ventricular segment there is another from which the ventricles develop.

The thick-walled ventricles are developed for the purpose of keeping up a continuous supply of blood to the tissues, and therefore their primary function is to contract forcibly.

It used to be taught that the thin-walled auricles acted as reservoirs in which blood collected during contraction of the ventricles, so that it might be supplied to them rapidly during relaxation, by contraction of the auricles.

It has recently been shown that the auricles throw only a small quantity of blood into the ventricles, and that the chief function of their short, sharp contraction is the closure of the auriculo-ventricular valves. This closure is normally effected by the sudden projection of blood from auricle to ventricle producing a partial vacuum on the auricular side of the valve, which draws their cusps together, beginning at their attached and ending at their free margins. By this mechanism regurgitation of blood from ventricle to auricle during the commencement of ventricular systole is prevented.

Yandell Henderson and F. E. Johnson, *Heart*, vol. iv., 69-82.

We should expect to find in the remains of the primitive cardiac tube, not the propulsive function of contractility, but the regulating functions of stimulus production, excitability, and conductivity.

By ligaturing the heart at different levels we have just observed that the most excitable part of the heart is the venous end; consequently we should expect to find the maximum excitability in that part of the primitive remains of the cardiac tube which is nearest to the veins—*i.e.*, at the sino-auricular node. What proof is there in support of this?

"Lectures on the Heart," by T. Lewis, 1916.

To answer this question, we must turn to the researches of Lewis and of Einthoven, the inventor of the string galvanometer.

If a point in a sheet of muscle is stimulated, and the muscle is explored by two non-polarisable electrodes connected to the string galvanometer, one electrode being placed near the point of stimulation, the other rotated through an angle of 90°, it is found that the largest excursion of the galvanometer is always given when the electrodes are arranged radially to the point of stimulation. A little consideration will show that this must necessarily be so, because the space of time which elapses while the excitation wave passes from one electrode to the other is always greatest when the electrodes are in a radial position. Working upon this principle Lewis explored the auricle of the dog, and was able to locate the site at which the stimulus to contraction was first manifested (*i.e.*, the place of maximum excitability) as being in the sino-auricular node. This was further proved by the following experiments:

1. If one electrode is placed over the origin of the excitation wave, and the other upon a circle of points around it, then the galvanometer deflections will all be good, because the electrodes in each lead are in a radial position to the point of origin of the excitation wave. Further, all the deflections will have the same sign because at the beginning of the excitation wave the muscle under the central electrode is always relatively negative to the muscle under the other electrode. By applying this principle to the auricular wall of the dog, it was found

that the only position which gave these electrical reactions was that in which the central electrode was over the sino-auricular node.

2. It is only when one of the two electrodes lies directly over the site at which the excitation wave begins, that all the galvanometer deflections are "intrinsic," and that there are no "initial" extrinsic deflections.

On exploring the auricular wall, the only position which gave no initial extrinsic deflection was found to be that in which one electrode was placed over the *s–a* node.

3. The same lead from a normal heart always gives the same electric curve. If the stimulus for contraction arises from some point other than the normal, an abnormal electrocardiogram is given.

Lewis applied this principle as follows: Taking a normal electrocardiogram from the dog, and using the same leads, he found that when the auricular wall was stimulated at different points, an electrocardiogram of similar outline to the normal one was obtained only when the auricle was stimulated in the neighbourhood of the S–A node.

We are therefore fully justified in regarding the S–A node of Keith and Flack as the point of maximum excitability in the heart. In pathological conditions, such as premature contractions, auricular flutter, and fibrillation, other parts of the auricular, or even the ventricular, wall become more excitable than the S–A node, with the result that the cardiac contraction begins at these abnormal foci.

It appears from recent work that increased excitability is associated with increased permeability of the cell membrane.

In the quiescent state the membrane of the muscle cell is regarded as being semipermeable—*i.e.*, permeable to one ion, but not to the other, forming what is known as a Helmholtz double layer. In a state of excitation there is an increase in the permeability of the muscle cell; the cell membrane now becomes permeable to both ions, the state of polarisation of the Helmholtz double layer undergoing depolarisation, and between the active and non-active part of the muscle there is a difference of potential which can be recorded by the galvanometer. Lillie records experiments in which substances such as bile salts, saponin, and sodium oleate, which are known to make the cell membrane more permeable, cause quick, vigorous twitches of frog's muscle.

Amer. Journ. Physiol., xxviii., 197-222.

Siebeck has shown that potassium chlorate enters more quickly into excited muscles than into resting ones, facts which indicate increased permeability during stimulation.

Pflüger's Arch., cl., 316-324.

Lillie showed in 1911 that the critical change in stimulation is a rapid increase in the ionic permeability of the cell membrane. Nernst's hypothesis is that stimulation requires a certain critical increase in the concentration of ions at the membrane; but an increased ionic permeability gives rise to a depolarisation at the membrane; and, since this depolarisation effect invariably accompanies stimulation, it must be regarded as an essential feature of the process. From this reasoning it is evident that any increased concentration of ions at the cell membrane which is to produce stimulation must be of such a character as to "decrease" the already existing physiological polarisation.

Amer. Journ. Physiol., 1911, xxviii, 197.

Therefore a current which is passed through an irritable tissue in such a direction as to increase the physiological polarisation of the cell membrane will not effect stimulation, but when the current is broken stimulation will occur, as the polarisation will suddenly be diminished.

Theoretically an irritable tissue at the height of stimulation should be unaffected by the passage of an electric current, since the cell membranes are freely permeable to its ions and the current therefore can have neither a polarising nor a depolarising effect. The truth of this hypothesis is shown by the refractory period. It is significant that the refractory period has approximately the same duration as the electrical variation—*i.e.*, as the period of increased ionic permeability.

Tait divides the refractory period into two parts: (*a*) the absolute, (*b*) the relative. He suggests that the absolute refractory period corresponds to the negative phase of the electrical curve of active muscle, which, he says, is disintegrative or katabolic, whilst the relative refractory period corresponds to the positive return of the electrical curve, which he regards as reintegrative or anabolic.

Tait, *Quarterly Journ. of Experimental Physiol.*, 1910, p. 221

3. The Function of Conductivity.

Before Gaskell began his work in 1881 the sequence of the contraction of the heart chambers and the time of opening and closing of the valves had been accurately established by recording the changes of blood-pressure within the auricle and ventricle and between the ventricle and aorta by the instruments already described.

It was thought that the sequence of the contraction of these chambers was due to the stimulus starting in Remak's ganglion cells and that it was conveyed by the fibres of these nerve cells throughout the auricle and ventricle. The fact that the ventricles contract after the auricles was explained as being due to a delay in the passage of the stimulus at the relay centre—*i.e.*, in Bidder's ganglion cells. It was observed that in the dying heart of a frog the ventricles beat at a slower rate than the auricles; this was also regarded as due to a disturbance in these cells.

Gaskell showed that the Remak and Bidder's ganglion cells can be removed without causing any disturbance in the rhythm. In the heart of the tortoise the coronary nerve which supplies the ventricle is more accessible than in the frog; after division of this nerve contraction still passes from auricle to ventricle. By using his suspension method, instead of the endocardial blood-pressure apparatus, Gaskell was able to record the contraction of the chambers after a number of interdigitating cuts had been made across the auricle, cuts so arranged that all the nerves were cut across at least once; yet the sequence of the contraction of the chambers still remained normal.

Journ. Physiol., 1883, vol. iv.

Further, experimenting on the heart of the tortoise after cutting all the nerves, he divided the auricle across so as to leave only a narrow bridge of muscle (free from nerves) between auricle and ventricle. By clamping this bridge of muscle, or cutting it still narrower, he found that there was a delay in the stimulus reaching the ventricle, or that the ventricle responded not to each auricular contraction but only to every second, third, or fourth auricular contraction.

If the muscular bridge is cut right across, but the coronary nerve left, the ventricle ceases to beat; but it soon begins again, at a slower rate and independently of the auricle. If the coronary nerve be cut across and the bridge of the muscle left intact, the rate of ventricular contraction is dependent upon the stimulus derived from the auricle. By these experiments Gaskell proved that the stimulus is conducted from auricle to ventricle by the muscle cells, not by nerves. Engelmann later carried out experiments on similar lines and came to the same conclusion.

Pflüger's Arch., 1882, vol. xxix.

It was not, however, until 1893, when His discovered the *a–v* bundle, that the muscular connection between auricles and ventricles in the mammalian heart was demonstrated. In 1894 His produced complete heart block experimentally; and by graduated compression of the *a–v* bundle in the dog, Erlanger, working at the suggestion of Osler, was able to produce 2 to 1, 3 to 1, and 4 to 1 rhythms, finally passing into complete heart block. At an earlier date Gaskell had produced similar results by compressing the *a–v* junction, in his conduction experiments on the heart of the frog and tortoise.

Wiener Med. Blätter, 1894, No. 44. *Journ. of Experimental Med.,* 1906, vol. viii., p. 8. *Journ. Physiol.,* vol. iv., 112.

The conduction of the stimulus by the *a–v* bundle from auricle to ventricle was therefore proved before the time of the invention of the string galvanometer by Einthoven; but only by the aid of this instrument has the conduction of the stimulus been followed throughout its course—*i.e.,* starting at the *s–a* node, spreading through the walls of the auricles, passing along the *a–v* bundle to the ventricles, and then along its right and left branches to be finally distributed to the musculature of the ventricles through the subendocardial network known as the Purkinje system.

With the aid of the string galvanometer we will now consider the more recent experimental work on conduction of the excitation wave. Before using the galvanometer for this purpose it is important to be quite clear that the electrical changes it records are not the result of muscular contraction, but are due to a passage of a wave of excitation.

I have already mentioned, in considering the final deflection T in the ventricular complex of the galvanometer curve, that Mines had proved that the excitation process giving an electrical curve is possible without the contraction process. His demonstration consisted of perfusing a frog's heart with Ringer's solution from which the calcium salts were omitted, when, although no con-

Journ. Physiol., 1913, xlvi., p. 188, 235 traction took place, a normal electric curve was recorded by the galvanometer. With reference to the part played by calcium salts in muscular contraction Locke concludes that "calcium is necessary for the conversion of the heart's chemical energy into the mechanical energy of its beats."

Journn. Physiol., 1913, xli.

By applying the methods I mentioned under the location of the normal stimulus in the *s–a* node, and also by taking a series of contacts along a straight line arranged radially to the *s–a* node, and by noting the time at which the excitation wave arrived at the several contacts, and that it always reached a contact proximal to the *s–a* node before it reached a more distal one, Lewis demonstrated that the normal stimulus arises not only in the *s–a* node, but passes also from the node in a radiating manner all over the auricular wall and into the roots of the great vessels. He likens the spread of the excitation wave throughout the auricle to the spread of a fluid upon a flat surface. The rate of transmission of the stimulus through the auricular musculature is about 1,000 mm. per second.

"Lectures on the Heart," by T. Lewis, 1916.

There is no evidence that the excitation wave passes down the septum to the *a–v* node at a greater rate than it travels over the other walls of the auricles. Lewis and his co-workers point out how advantageously the *s–a* node is situated in order to transmit the stimulus through the auricle; for in the mammalian heart all the muscle fibres of the auricle collect in a fan-shaped manner to a "concentration point" as he terms it, just below the *s–a* node. The actual conduction of the stimulus is due to a depolarisation, and an increase in the ionic permeability, of the limiting membrane of adjoining cells.

Turning now to the conduction of the stimulus in the ventricle, we find that Gotch declares that the electrical changes in the heart are caused by the passing of the excitation process as a wave over the ventricles; starting at the junction of auricles and ventricles, the excitation wave continues down to the apex and then back to the aortic part of the base. It thus passes, according to Gotch, through the ventricle in the order of its tubular development, appearing twice at the base but only once at the apex in each ventricular systole.

"The Succession of Events in the Contracting Ventricle as shown by Electrometer Records," *Heart*, 1909-10, i. 235-261.

By using the string galvanometer and adopting a very accurate method of measurement of time relationship, Lewis has disproved Gotch's hypothesis. He finds the rate of conduction of the Purkinje system is about 2,000 mm. per second, whilst the rate of conduction of the ventricular muscle fibres is about 400 mm. per second, or only one-fifth as fast as the rate in the Purkinje system. Lewis appears to have proved that the excitation wave in the ventricle does not pass from muscle fibre to muscle fibre of the mass of the ventricular musculature, but is first conducted by the right and left branch of the *a–v* bundle to the Purkinje system, by which it is distributed over the whole subendocardial surface of both ventricles. The stimulus, which passes in a clockwise direction over the right ventricle and anticlockwise over the left, is conducted so rapidly by the Purkinje system that it appears almost simultaneously over the whole subendocardial surface. The varying rate of appearance of the excitation wave over the "superficial" surface of the ventricle is due to the varying thickness of the muscle.

Purkinje, "De Musculari Cordis Structura."

In 1839, in "De Musculari Cordis Structura," Purkinje first described those subendocardial cells which are now called by his name. Since then there has been much discussion and difference of opinion as to whether the cardiac muscle consists of a number of uninucleated branched cell fibres, or of a continuous protoplasmic mass containing nuclei at intervals, with the muscle fibrils in complete continuity throughout—that is to say, forming a complete syncytium, or one single large contractile mass. The former view is supported by Schweigger-Seidel, on the ground that when certain staining methods are used there is evidence of distinct septa across the fibres, and also that the fibres, when treated with certain reagents, tend to separate into branched uninucleated pieces.

Stricker's "Handb. d. Gewebelehre," 1869.

But the appearance of dead cells which have been stained or treated with certain reagents is inconclusive evidence as to the shape and behaviour of the living cells.

If the Purkinje system, which is very well developed in the sheep and ox, be examined under the microscope, it is found to consist not of a number of uni- or bi-nucleated cells, but of a complete syncytium of protoplasm in which are developed cross-striated fibrils, at points equidistant from the nuclei.

There is also evidence to show that the cardiac muscle fibre is only a further development of the cross-striated fibrils within the syncytium of the Purkinje system. The function of the fully developed muscle fibres is to contract and force blood into the arteries; whilst the function of the Purkinje system is the almost simultaneous conduction of the stimulus all over the endocardial surface of the ventricular musculature.

Mines, by the aid of his special perfusion apparatus, has carried out researches into the effect on the heart-beat of H ion concentration. Amongst other results obtained he has shown that if the heart be perfused with an acid solution the *a–v* interval is first lengthened; then the ventricle fails to respond to every auricular contraction; and, finally, complete heart block sets in. Perfusion with an alkaline solution quickly removes the heart block, and the *a–v* interval is then gradually shortened.

Journ. Physiol., 1913, xlvi.

4. THE FUNCTION OF CONTRACTILITY.

Besides possessing a long refractory period which we have already described, Bowditch, in 1871, drew attention to another property of cardiac muscle. If cardiac muscle contracts at all in response to a stimulus, it does so with its full force—*i.e.*, " all or nothing "—wherein it differs from skeletal muscle. Gotch and Keith Lucas have shown that the reason why contractions of skeletal muscle are proportional to the stimulus is because fewer fibres contract in response to weaker stimuli than to stronger ones, the fibres not being functionally continuous. The cardiac muscle fibres, on the other hand, having developed from a complete syncytium, are all functionally continuous, so that a contraction of one fibre involves a contraction of all.

Ber. d. sach. Gesellsch., 1871, S. 687

Starling has recently thrown fresh light on the function of contractility of the ventricular muscle. In his Linacre Lecture at Cambridge (1915), he described the results of his experiments with the " heart-lung preparation." By an ingenious apparatus he is able to control the amount, temperature, and pressure, of the blood that enters the heart, and also the peripheral resistance. He is able, further, to measure directly the blood-pressure in the aorta, and the output of the heart, also the flow of blood through the heart muscle itself by way of the coronary vessels.

"The Linacre Lectures on the Law of the Heart," by Ernest H. Starling. Cambridge, 1915.

Starling finds that, provided the inflow to the heart be constant, the output remains the same, irrespective of the aortic blood-pressure. This statement is only valid when the aortic pressure is within physiological limits—*i.e.*, between 44 and 208 mm. of Hg. Although his experiments are so striking that I quote the table giving his figures and conclusions, I wish to point out that it is doubtful how much importance can be placed upon his conclusions because in the experiment the heart was working under abnormal conditions, the blood circulating through the heart not only was poisoned but lacked the internal secretions of the ductless glands.

Arterial Pressure, mm.·Hg.	*Systemic Output, c.c. per Min.*	*Total Coronary Output (calculated).*	*Total Output of Left Heart.*	*Venous Pressure, cm.* H_2O.
84	811	40·80	851·80	9·6–12·4
108	790	50·30	840·30	8·8–12·0
140	770	70·75	840·76	8·0–11·2
170	750	117·40	867·40	8·0–10·8
208	600	260·30	860·30	12·0–22·0
104	760	80·30	840·00	8·4–10·4
104	750	80·30	830·00	8·0–10·0
44	790	30·40	820·00	7·6–10·0

Inflow of blood into the heart was maintained constant.

“ Two important facts are rendered evident by these results. In the first place, provided the inflow remains constant, it seems to be immaterial to the heart whether it has to contract against a resistance of 44 mm. Hg, or 208 mm. Hg. In each case it puts out as much blood as it receives, so that the total outflow remains constant whatever the arterial pressure. Of course there is a limit which must not be exceeded. If the pressure is placed too high, say at 200 to 220 mm. Hg, the heart may fail, and become distended with blood which it is unable to expel. Within physiological limits, however, we may say that the output of the heart is independent of the arterial resistance, so that whatever work is given the heart to do it can do it without fail. The second fact which results from this experiment is that the fraction of the total output of blood which passes through the coronary vessels rises steadily with the height of the arterial pressure.

This means that the more work the heart has to do the better it is supplied with blood—*i.e.*, with the oxygen and nutriment necessary to furnish it with energy."

If, on the other hand, the arterial pressure be kept constant and the inflow increased, then the outflow from the heart is increased to the same degree. Krogh has shown that the output of the heart may vary as much as from 3 to 21 litres per minute.

By recording the intraventricular pressure, and working on the principle that when the heart is in a plethysmograph any variation recorded in its volume must indicate a shortening or lengthening of the individual muscle fibres, Starling obtained the following information: during diastole of the ventricle the inflowing blood does not promote any tension in the muscle fibres, since these fibres lengthen to accommodate the incoming blood; and the force of the resulting contraction is proportional to the length of the muscle fibres.

Beyond a certain point any further increase in the inflow of blood during ventricular diastole will begin to set up tension in the fibres; at this point and beyond the muscle contracts at a disadvantage, the force of contraction diminishes, and heart failure sets in. Seeing that a lengthening of muscle fibre is necessary to an increased force of contraction, a little consideration will show how increased contraction is obtained when there is a sudden increase of arterial pressure, but a constant inflow from the veins. Immediately after the rise of arterial pressure the ventricular fibres will not be long enough to develop sufficient force to empty the whole contents of the ventricle into the aorta; during the next diastole the ventricular cavity will contain this residual blood plus the usual supply from the veins, and so its fibres are increased in length and the next contraction is correspondingly stronger than the last. In a few beats enough residual blood will have accumulated in the ventricle to cause the muscle fibres to increase in length sufficient to give the force of contraction necessary for the ventricle to send out into the arteries as much blood as it receives from the veins. Increased arterial pressure means also an increased flow of blood through the coronary arteries to the heart muscle, with a resulting increase in nutrition, so that the physiological condition of each muscle fibre is improved until it can develop so great an energy of contraction that the fibres may now shorten, and the heart return to its normal size, its chambers being completely emptied, although against the increased arterial pressure. When the muscle is not healthy, or the demand upon the heart too heavy, then the ventricles do not return to their normal size, but remain dilated, since it is only in this condition that they are able to develop the necessary force of contraction. Then residual blood may continue to accumulate in the ventricles after each contraction; in this case dilatation is progressive and heart failure sets in.

Journ. Physiol., 1914, vol. xlix., p. 378.

Lovatt Evans has shown that the metabolic changes in the heart are increased in proportion to the increase in the work the heart has to do.

"Linacre Lecture," 1915.

Starling says: "The law of the heart is thus the same as the law of muscle tissue generally, that *the energy of contraction, however measured, is a function of the length of the muscle fibres*. . . .

"Now, the effect of lengthening the muscle will be to increase the extent of the active surface so that we may restate our general proposition in the following way: *Any increase in the extent of active surface increases the energy of change.*"

He regards the active shortening of muscle fibres—that is, the force of contraction as due to the conversion of chemical energy into surface energy.

This wonderful adaptation of the heart to the varying demands made upon it is equally well carried out after the destruction of all the nerves which connect the heart to the central nervous system.

It is well known that the fibrous pericardium acts, not only as a lubricated surface to diminish the friction during the movement of the heart, but limits also the degree of dilatation during diastole. If the pericardium be slit open the heart tends to expand through the opening during the diastolic period. Claude Bernard showed further that if the pericardium be freely cut away the tricuspid valve ceases to be entirely efficient. He believed that the efficient closure of this valve is in some way dependent upon the support of the fibrous pericardium.

Considering what has already been written on the conduction of the excitation wave, and also the law that the galvanometer deflections depend upon the point of origin and direction of the excitation wave, the relative positions of the leads, and the space of time which elapses while the excitation wave passes from one electrode to the other, it will be seen that the degree of deflection in an electrocardiogram is in no way a measure of the strength of the cardiac contraction; and therefore the extent of the galvanometer deflections cannot give any information as to the power of the heart muscle.

5. THE FUNCTION OF TONICITY.

It is usual to define muscular tone as the power to retain a certain amount of contraction even when active movement has ceased. We have seen, however, that the length of the ventricular muscle fibres depends upon the quantity of blood entering this chamber. It is therefore very difficult to define tone exactly. Starling describes the tone of the heart muscle as synonymous with its physiological condition, or the fitness of its fibres. The physiological condition of the heart muscle is measured by the pressure that is developed within the ventricles during a contraction, starting with the muscle fibres at a certain initial length; that is to say, its tone is measured by the energy set free per unit length of muscle fibre at each contraction.

CARDIAC IRREGULARITIES AND ABNORMAL RHYTHMS.

Let us now consider the various forms of cardiac irregularity and abnormal rhythms.

When confronted with the problem of a venous tracing, or an electrocardiogram, it is necessary to be familiar with all the possible conditions it may signify. If the curve is not immediately easy of solution, all the various conditions that might be represented must be gone through systematically, in order to determine the exact signification. I give, therefore, in skeleton form, a classification of all the various forms of cardiac irregularity and abnormal rhythm which are always before my mind when attempting to solve a difficult tracing, and I make a habit of working through this list systematically, eliminating each condition in turn.

1. Sinus, respiratory, or youthful type of irregularity.
2. Extra systoles or premature contractions
    - (*a*) Ventricular varieties of.
        - (1) The ventricular contraction occurs before the normal auricular contraction.
        - (2) The ventricular contraction occurs at the same time as the normal auricular contraction.
        - (3) The ventricular contraction follows the auricular at too short a distance for the contraction to be the result of an auricular stimulus.
        - (4) Interpolated—*i.e.*, a ventricular systole occurs between two normal ventricular contractions.
        - (5) Multiple ventricular extra systoles.
    - (*b*) Auricular varieties of.
        - (1) The ventricular contraction has the normal relation to the auricular, but the auricular systole is premature.
        - (2) Multiple auricular extra systoles.
        - (3) Interpolated auricular extra systoles.
        - (4) Blocked auricular extra-systoles—*i.e.*, a premature auricular systole which fails to stimulate the ventricle to contract.
    - (*c*) Nodal.
        - Auricle and ventricle contract together before the normal auricular contraction is due.
3. Simple paroxysmal tachycardia
    - (*a*) Auricular.
    - (*b*) Ventricular.
    - (*c*) Nodal.
4. Auricular flutter.
5. Nodal rhythm.
6. Auricular fibrillation.
7. Heart block—partial or complete.
8. Pulsus alternans or exhaustion of the function of contractility.
9. Sino-auricular block—*i.e.*, the stimulus at the sino-auricular node fails to give rise to a contraction, so that a complete cardiac cycle is missed.
10. Escaped ventricular beats. In cases of very slow rhythm (for example, strong vagal tone, or partial heart block) the ventricle occasionally contracts on its own account before the delayed stimulus from the auricle reaches it.

“Clinical Disorders of the Heart-Beat,” by Thos. Lewis.

Let us now consider each condition separately. After giving a brief note of its cause, each will be illustrated by polygraphic and electrocardiographic tracings; then a short note will be given of how to diagnose the condition clinically, without instrumental aid. Since there is a fashion amongst the less enlightened to cast ridicule upon these instruments, it is necessary to insist once more upon the truth that it was only through the aid of the clinical polygraph and the string galvanometer that order was introduced into the hopeless confusion of cardiac irregularities and abnormal rhythms before these instruments were invented, and how by them abnormal rhythms have been put in their proper place with clear understanding of their cause and mechanism.

1. Vagal, Sinus, Respiratory, or Youthful Type of Irregularity.

This irregularity is due to a variation in vagal tone and a consequent waxing and waning of stimulus production or excitability.

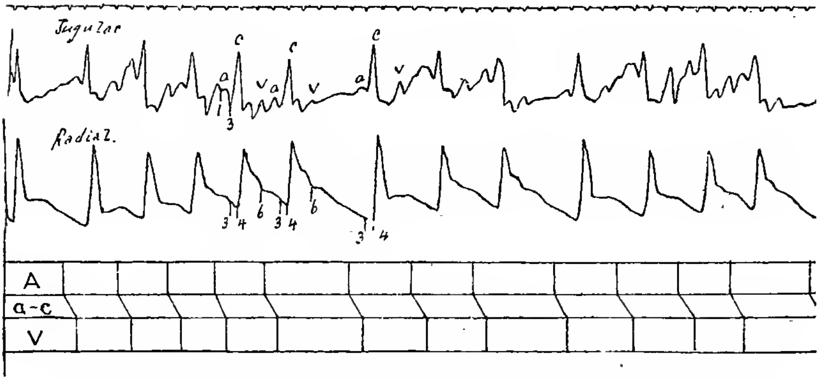


Fig. 30.—Simultaneous Tracings of the Jugular and Radial Pulses, showing the Sinus or Vagal Type of Irregularity.

There is no evidence of auricular or ventricular contraction during the long pauses; this distinguishes the vagal form of irregularity from that due to extra systoles.

Fig. 30, which was taken from a soldier, is a good example of sinus arrhythmia.

This is an interesting case, because the man was sent to hospital with a note from his medical officer to the effect that he should be discharged from the army on account of the irregularity of his pulse. The doctor must therefore have regarded this irregularity as significant of gross cardiac disease. This opinion is not supported by experience; in fact, uncomplicated sinus irregularity is a sign of healthy heart muscle.

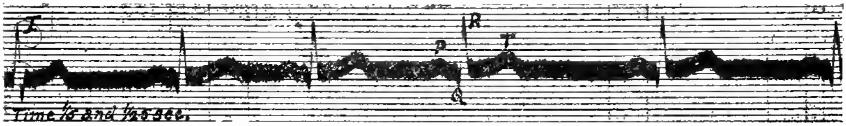


FIG. 31.—ELECTROCARDIOGRAM SHOWING THE SINUS OR VAGAL TYPE OF IRREGULARITY.

Fig. 31 is an electrocardiogram, showing the sinus or vagal type of arrhythmia.

Clinical Diagnosis.—This condition, as its name suggests, occurs in children and young adults. By making the subject breathe deeply a definite relation between the irregularity and the respiratory rhythm will be observed, the pulse-rate slowing during expiration. The heart sounds and apex pulsations correspond with the radial beats. The irregularity disappears when the rate of the heart is considerably increased by exertion, fever, or after an injection of atropine. The patient is not conscious of any irregularity, and the condition does not give rise to abnormal symptoms on exertion.

2. EXTRA SYSTOLES OR PREMATURE CONTRACTIONS.

This condition is due to some part of the auricular or ventricular wall becoming more excitable than the sino-auricular node, the normal pace-maker of the heart; the cardiac contraction beginning in this new excitable ectopic focus

It is important from the clinical point of view to learn that an increase in the function of excitability exposes the heart to the liability of having to contract in response to stimuli which under normal conditions are too weak to evoke a contraction.

In describing extra systoles it is convenient to refer to the combined pulse spaces of the extra systole and the preceding beat as the "irregular" period.

(*a*) *Ventricular Extra Systoles.*

In Figs. 32 and 33, which are examples of ventricular extra systoles, the premature contractions are marked X.

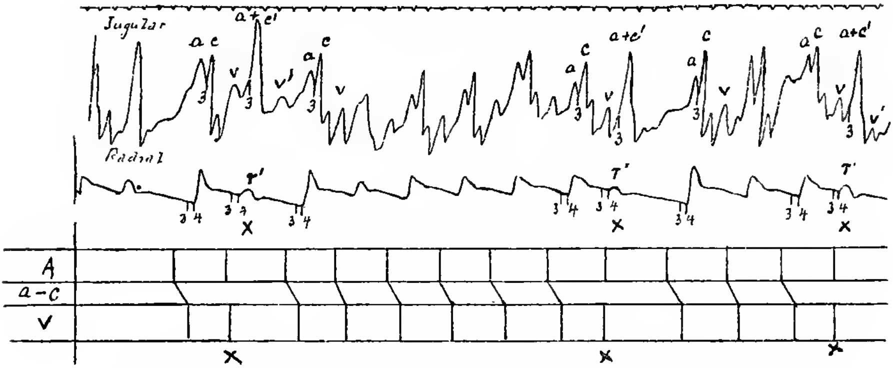


Fig. 32.—Simultaneous Tracings of the Jugular and Radial Pulses, showing Some Ventricular Extra Systoles marked X.

The key diagram represents the condition very plainly. The down strokes in the space A represents the beginning of the auricular waves *a* in the jugular tracing; and the down strokes in the space V represents the beginning of the carotid waves *c*; and the slanting lines connecting them represent the *a–c* interval.

It will be seen from the key diagram that the pause following the premature beat is fully compensatory, as the irregular period is equal to that of two normal contractions. In Fig. 32 the irregular period, due to some sinus irregularity, is slightly greater than that of two normal pulse periods.

The large " steeples " in the venous tracing at the time of the extra systoles are due to forcing of the blood into the veins instead of into the ventricle during auricular contraction, as both auricle and ventricle are in contraction at the same time.

In both tracings there is some slight sinus irregularity.

With the aid of the electrocardiogram we are able to say in which ventricle a premature contraction originates.

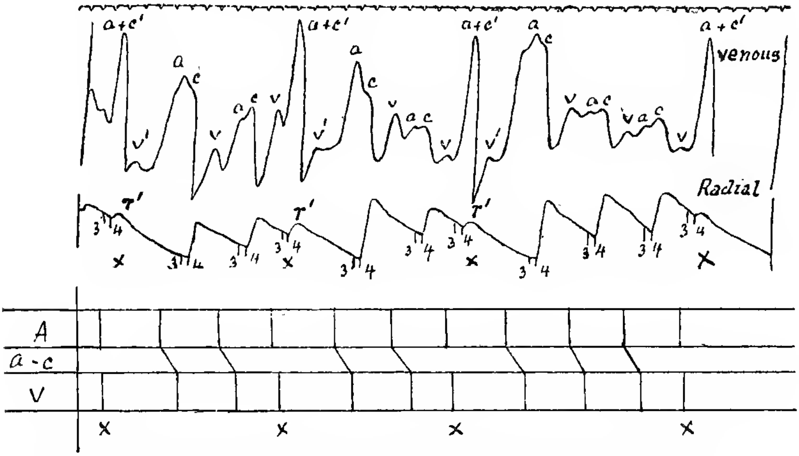


Fig. 33.—Simultaneous Venous and Radial Tracings showing Ventricular Extra Systoles marked X.

A key diagram is placed below the radial tracing.

The stimulus which causes this form of ectopic contraction does not reach the ventricle by way of the junctional tissue, but originates in its musculature; therefore the ventricular complex will be abnormal. It has a similar outline to that of a bundle branch lesion. The deflection caused by a right or left ventricular extra systole corresponds in outline to that of defective conduction of the left or right branch respectively of the *a–v* bundle. The deflections in lead I. are usually in the opposite direction to those in lead III.; lead II. is uncertain—it usually follows lead III. A right ventricular extra systole occurring in lead I. is shown in Fig. 34, and in lead III. in Fig. 35. A left ventricular extra systole occurring in lead I. is shown in Fig. 36 and in lead III. in Fig. 37.

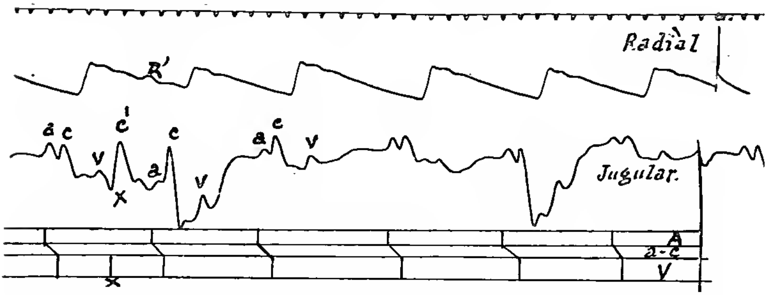


FIG. 38.—SIMULTANEOUS TRACINGS OF JUGULAR AND RADIAL PULSES, WITH KEY DIAGRAM INSERTED, SHOWING AN INTERPOLATED VENTRICULAR EXTRA SYSTOLE AT X, MARKED *c′* IN THE JUGULAR TRACING, AND R′ IN THE RADIAL TRACING.

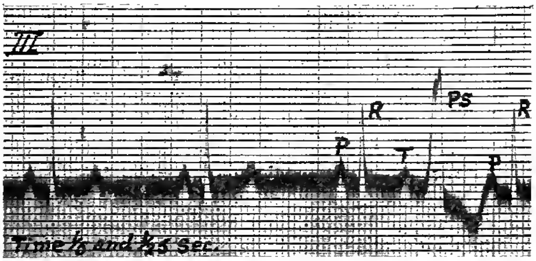


FIG. 39.—ELECTROCARDIOGRAM SHOWING AN INTERPOLATED VENTRICULAR EXTRA SYSTOLE P.S. IN LEAD III.

It originates in the right ventricle.

A premature ventricular contraction which occurs during a normal diastolic period and between two normal ventricular systoles is known as an interpolated ventricular extra systole. An example of this variety of ventricular contraction is shown in the polygraphic tracing, Fig. 38, and the electrocardiogram, Fig. 39.

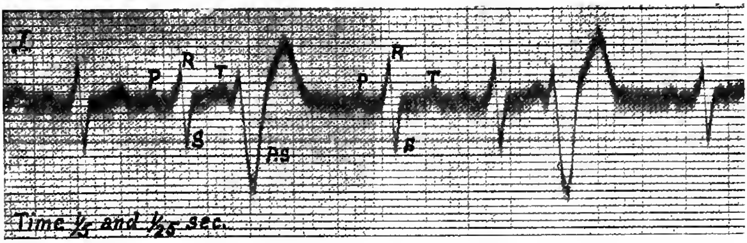


FIG. 34.—ELECTROCARDIOGRAM SHOWING THE OUTLINE OF A RIGHT VENTRICULAR EXTRA SYSTOLE, P.S., IN LEAD I.

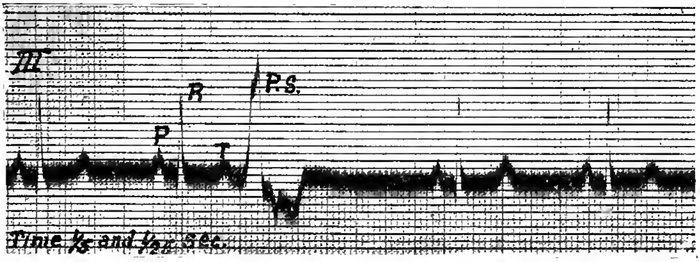


FIG. 35.—ELECTROCARDIOGRAM SHOWING THE OUTLINE OF A RIGHT VENTRICULAR EXTRA SYSTOLE, P.S., IN LEAD III.

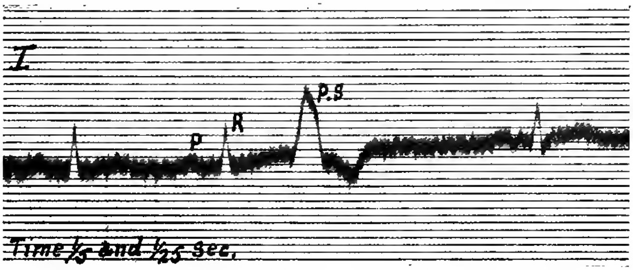


FIG. 36—ELECTROCARDIOGRAM SHOWING THE OUTLINE OF A LEFT VENTRICULAR EXTRA SYSTOLE, P.S., IN LEAD I.

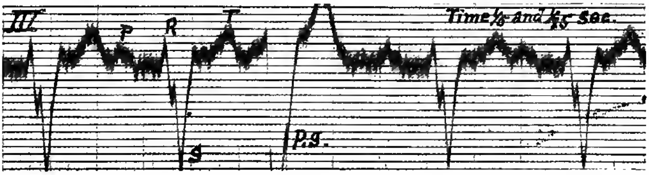


FIG. 37.—ELECTROCARDIOGRAM SHOWING THE OUTLINE OF A LEFT VENTRICULAR EXTRA SYSTOLE, P.S., IN LEAD III.

(*b*) *Auricular Extra Systoles.*

An example of auricular extra systoles is shown in the polygraphic tracing, Fig. 40.

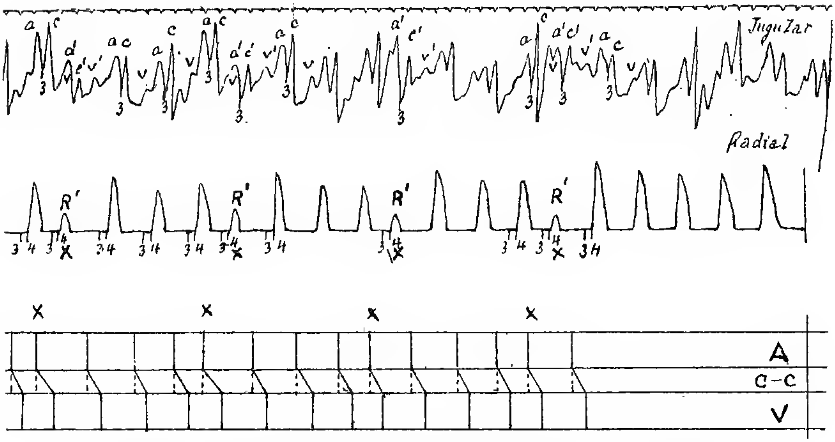


Fig. 40.—Simultaneous Jugular and Radial Tracings with Key Diagram, showing Auricular Extra Systoles, marked *a′* in the Venous Tracing, R′ in the Radial Tracing, and X in the Key Diagram.

The key diagram shows that the pause following the extra systole is not fully compensatory, the irregular period being less than that of two normal beats.

The increase in the *a–c* interval during the extra systole, which is well shown in the key diagram, is due to the stimulus from the premature auricular contraction arriving at the *a–v* bundle before its conductivity has fully recovered from the passage of the preceding stimulus.

This tracing shows very well the increase in the size of the pulse wave that follows the extra systole. This is readily explained by the axiom that the size of the contraction depends upon the length of the muscle fibres. As the feeble contraction of the premature beat does not empty the ventricle, and as the diastolic period following the extra systole is longer than normal, the ventricular cavity will contain an abnormally large quantity of blood at the beginning of the next systole; this increases the length of the muscle fibres, and therefore results in a more powerful contraction. Further, since the premature contraction forces only a small quantity of blood into the aorta, the arterial pressure at the time of the following ventricular systole will be below normal.

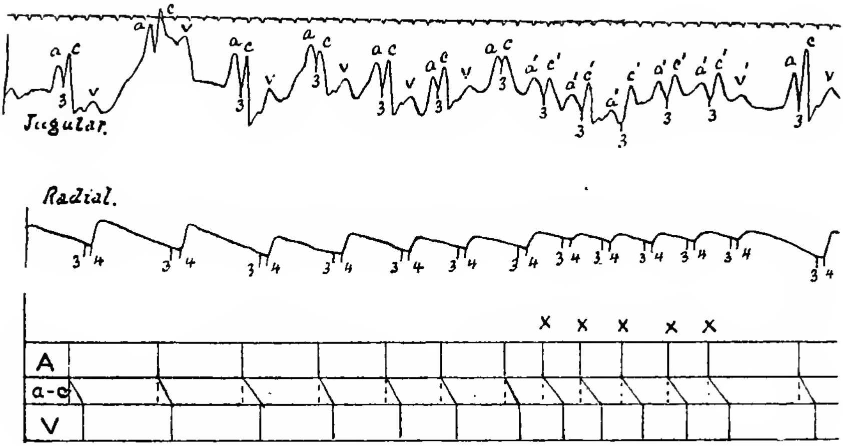


Fig. 41.—Simultaneous Tracings of the Jugular and Radial Pulses, with Key Diagram, showing Five Consecutive Auricular Extra Systoles.

Fig. 41 shows five auricular extra systoles occurring one after the other. During the auricular premature contractions the *a–c* interval is considerably increased, as we should expect. This tracing also shows some sinus arrhythmia.

In the electrocardiogram the ectopic contraction will be shown not only by the prematurity of the auricular complex, but also by inversion of the P wave when the stimulus originates outside the immediate vicinity of the sino-auricular node. The ventricular complex will be normal, because the excitatory process reaches the ventricles through the junctional tissue.

Fig. 42 shows two premature auricular systoles.

The Clinical Diagnosis of Premature Contractions or Extra Systoles.—The frequency of the occurrence of premature contractions increases with advance in years; they are less common in children, and become more common in the adult, whilst over the age of fifty they become comparatively frequent, especially under the influence of tea, coffee, tobacco, or (not a good sign) under the toxins of infections—*e.g.*, pneumonia. They are about twice as frequent in the male as in the female sex. They tend to disappear when the heart-rate is increased, as in pyrexia, and to become more frequent during the slow action of the heart shortly after exercise or exertion. As a rule, the patient is not aware of the premature systole, but is conscious of a void or feeling of oppression in the chest and complains of the shock against the chest wall caused by the strong contraction following the premature systole.

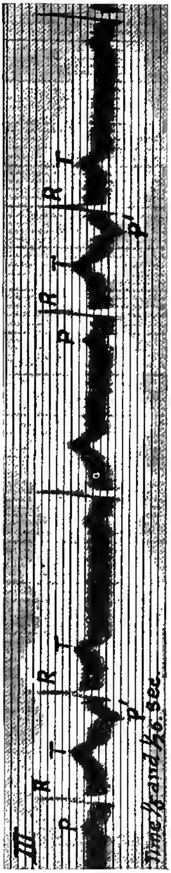


FIG. 42.—ELECTROCARDIOGRAM SHOWING TWO PREMATURE AURICULAR CONTRACTIONS, *p'* WITH INVERTED P WAVES.

The clinical signs of a premature systole will vary according to whether the aortic valves are opened or not. In the former case a small impulse will probably be felt in the radial artery, and two short, sharp sounds will be heard at the apex, followed by a compensatory pause. In the latter case a missed beat will be observed in the radial artery, and at the apex only one cardiac sound will be heard during this pause. Cardiac murmurs, when present, are modified by the premature contraction; an aortic systolic and diastolic murmur will occur only when the aortic valves are raised; a mitral systolic murmur is usually heard during the premature contraction, but it is shortened; a presystolic mitral murmur disappears if the auricles and ventricles contract at the same time.

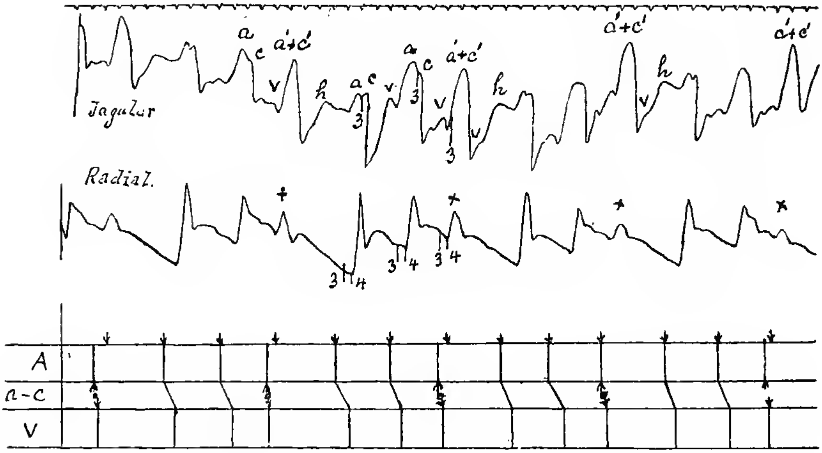


FIG. 43.—SIMULTANEOUS TRACINGS OF THE JUGULAR AND RADIAL PULSES, WITH KEY DIAGRAM.

The premature systoles are probably nodal in origin.

It is usually impossible to distinguish without a tracing between auricular and ventricular premature contractions, but the following points may be noted: If the extra systole be ventricular in origin and occurs at the same time as the auricular contraction, an abnormally large venous pulsation may be observed in the neck, caused by the auricle emptying its contents into the veins instead of into the ventricle; but this observation is not reliable, as an auricular premature systole may occur at the same time as the preceding ventricular contraction, when the same large venous pulsation will be observed. It is sometimes possible with the finger on the radial pulse to detect, in the case of ventricular premature beats, that the fundamental rhythm of the heart is not disturbed.

The first sound may be relatively exaggerated by the premature ventricular systole falling at the same time as the normal auricular contraction.

(*c*) *Nodal Extra Systoles.*

An example of nodal extra systoles following every second normal beat is shown in Fig. 43.

My reasons for regarding these premature beats as nodal and not ventricular are as follows: Both auricle and ventricle contract almost simultaneously and rather before the normal auricular contraction is due. If the extra systoles were ventricular they might bear the same relation to the auricular contractions as they do in this tracing, but we should expect the auricular contractions to occur at the normal time. The small arrows on the top line in the key diagram mark the time at which the normal auricular contractions are due. We must regard the stimulus as arising in the junctional tissue, possibly in the *a–v* node, and from there passing, on the one hand, up to the auricle, causing auricular contraction and exploding the stimulus material generated at the *s–a* node, and down, on the other hand, to the ventricle, where it gives rise to ventricular contraction. I have indicated this in the key diagram by a dot and two arrows in the *a–c* space. But I admit the above interpretation is open to criticism, and the tracing may prove to be one of ordinary ventricular extra systoles occurring at the same time as the normal auricular contractions.

It is impossible to distinguish clinically between nodal extra systoles and premature ventricular or auricular contractions.

3. SIMPLE PAROXYSMAL TACHYCARDIA.

This condition probably has the same origin as that of multiple premature auricular contractions. But instead of giving rise to isolated ectopic contractions the new focus at which the stimulus arises persists as the most excitable part of the auricle; with the result that the cardiac rhythm originates in this ectopic focus, instead of the sino-auricular node. The onset and offset of the paroxysm is sudden.

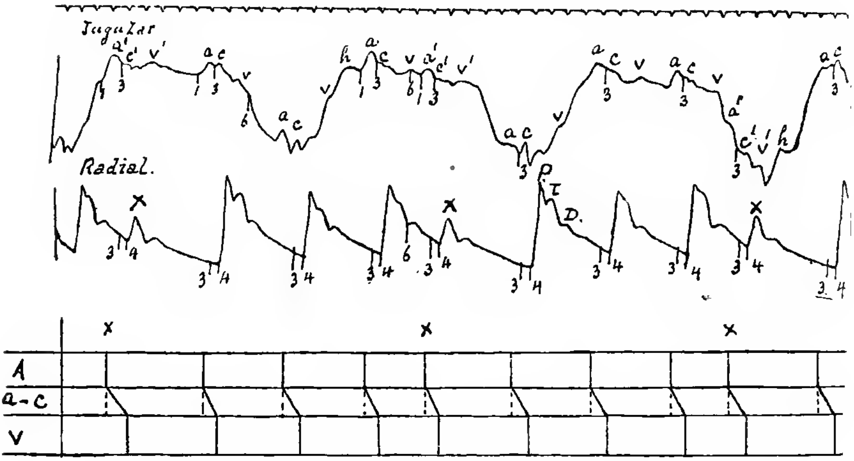


FIG. 44.—SIMULTANEOUS TRACINGS OF THE JUGULAR AND RADIAL PULSES, WITH KEY DIAGRAM, SHOWING THAT EACH FOURTH AURICULAR CONTRACTION IS PREMATURE.

This tracing was taken during a slow pulse period, from a man subject to attacks of simple paroxysmal tachycardia.

Figs. 44 to 47 were taken from a patient suffering from an attack of simple paroxysmal tachycardia. The tracing, Fig. 44, was taken when the heart was beating slowly. It shows that every fourth beat is an auricular

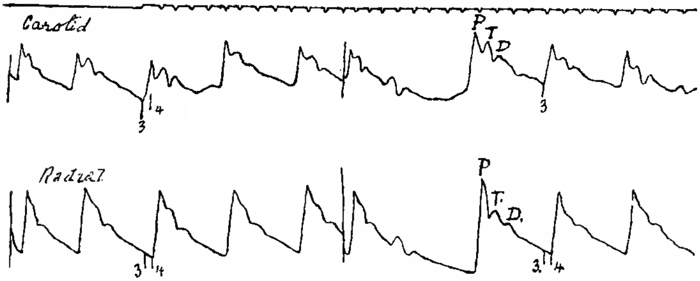


FIG. 45.—SIMULTANEOUS TRACINGS OF THE CAROTID AND RADIAL PULSES.

From the same patient as Fig. 44, 46 and 47.

extra systole. I have marked the premature beats X in both the radial tracing and the key diagram. The pause after the extra systole is not fully compensatory, the irregular period being a little less than two normal pulse spaces. The *a–c* interval is normal, being $\frac{1}{5}$ second

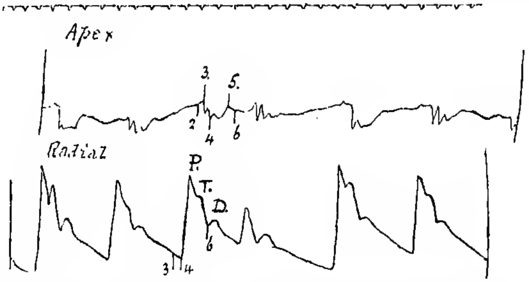
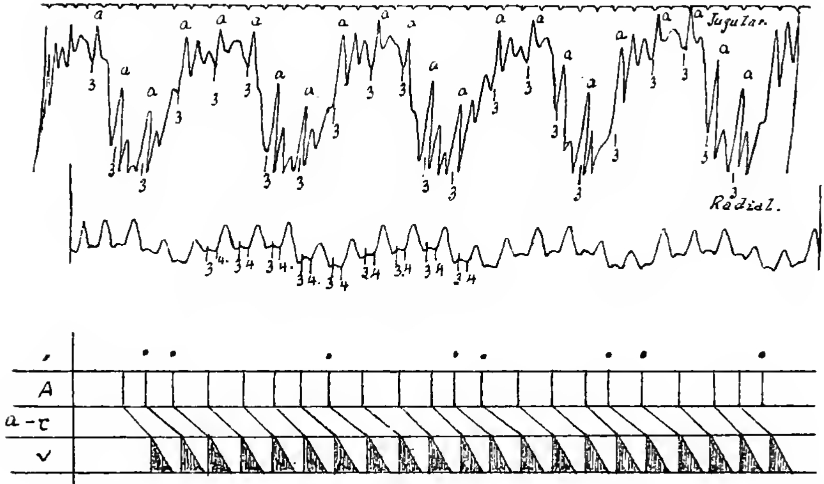


FIG. 46.—SIMULTANEOUS TRACINGS OF THE APEX BEAT AND RADIAL PULSE, SHOWING AN INVERTED CARDIOGRAM.

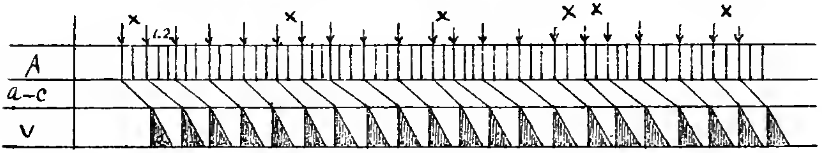
From the same patient as Figs. 44, 45 and 47.

Fig. 45 shows the carotid and radial pulse, and Fig. 46 the apex and radial pulse. The cardiogram in Fig. 46 is inverted and therefore not a true apex tracing, and hence what appears as the apex on palpation is really a part of the right ventricle.

As auricular extra systoles are frequent when the heart is beating slowly, I should suppose that the rapid rhythm is one either of simple paroxysmal tachycardia or of auricular flutter; but that in either case it is due to the ectopic focus, which is the origin of the stimulus causing the extra systoles, and which usurps the function of the *s–a* node as the pace-maker of the heart.



Key Diagram 1.



Key Diagram 2.

FIG. 47.—SIMULTANEOUS TRACINGS OF JUGULAR AND RADIAL PULSES WITH KEY DIAGRAMS.

For description see the text.

If we analyse the tracing, Fig. 47, we first notice that the radial pulse is regular and very rapid—*i.e.*, 176 per minute.

The jugular tracing shows a number of well-marked waves. Those I have marked *a* happen to occur practically at the same time at which the aortic valves open—*i.e.*, point 3; and these *a* waves are rather more prominent than the other waves. They are shown in the space A, key diagram 1; those marked with a dot occur a little before point 3. In the space V the ventricular contractions are represented by the downstrokes. Now the ventricular contraction may be in response to the preceding auricular contraction, but not to the one which lies directly above it; this gives an *a–c* interval of one and a half fifths second. While the heart is beating very rapidly we should expect the *a–c* interval to be increased, as conductivity of the *a–v* bundle has not time to completely recover before the next stimulus arrives; but according to key diagram 1 each auricular stimulus gets through, although it is delayed.

The next problem is to account for the waves between those marked *a* in the jugular tracing. Is the tracing one of auricular flutter in which only every third auricular stimulus gets through to the ventricle ? The answer is no, for in this case, since the radial pulse-rate is 176 per minute, the auricles would be contracting at a rate of 528 per minute, which I believe to be impossible. There must therefore be some other explanation.

Key diagram 2 is the same as key 1, with the addition of those waves which occur between the auricular contractions, the auricular contractions being indicated by an arrow. Between the second and third auricular contraction in key 2 I have marked these other waves, 1 and 2. When the auricle contracts the ventricle is in contraction also; at the cessation of auricular contraction the auricles relaxes, and the ventricle further expands the auricle by pulling down the auriculo-ventricular junction; this will cause a sudden fall in the jugular pressure shown by the sudden fall of the wave *a*. Now as the ventricle is still in contraction, this sudden relief of pressure on the auricular side of the tricuspid valve will, I think, cause a sudden movement of the cusps of the valve in the direction of the auricular cavity, and consequently a pressure wave through the auricle and great veins, giving rise to the wave marked 1.

The tricuspid valve will open early, owing on the one hand to the great congestion of the right auricle, and great veins, and on the other to the decrease of the intraventricular pressure due to the weakness of the contractions. When the tricuspid valve opens, the immediate rush of blood into the ventricle will momentarily close the valve, and give rise to the wave marked 2. In key 2 in the spaces marked X, there is only one wave instead of two between two auricular contractions. This is accounted for by early contraction of the auricle diminishing the interauricular space so that wave 2 is hidden in the following *a* wave. It is possible, however, that the waves between those marked *a* are instrumental. The tracing represents a case of simple paroxysmal tachycardia, the ventricle responding to each auricular contraction.

Looking at key 1 incidentally we see why, when the rhythm became rapid, the presystolic murmur which was present in the slow rhythm disappeared. Since the *a–c* interval is lengthened, auricular contraction occurs at the same time as the preceding ventricular contraction; hence no blood can pass from auricle to ventricle during auricular systole, and therefore no presystolic murmur can occur. These tracings have been included rather for the purpose of showing how difficult tracings should be tackled than as good examples of simple paroxysmal tachycardia. Fig. 47A is a more simple tracing of short paroxysms of tachycardia, showing the sudden onset and offset of the attacks. During a paroxysm the venous tracing shows only large waves, *a*, due to auricular contraction, there being no *c* or *v* waves.

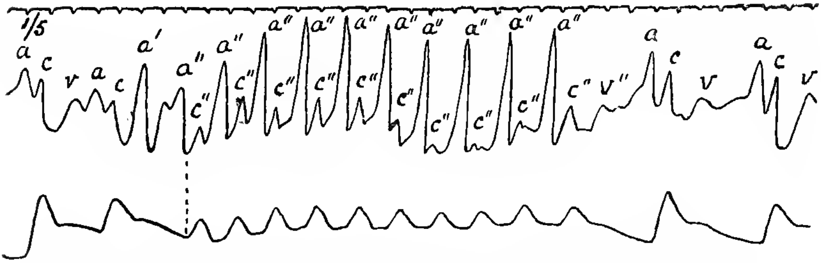


FIG. 47A.—RADIAL CURVE FROM A CASE OF PAROXYSMAL TACHYCARDIA, SHOWING A SHORT PAROXYSM, THE ONSET AND OFFSET OF WHICH WERE SIGNALLED BY THE PATIENT.

The slow periods are irregular. The pathological beats all arose at a single point in the auricular substance removed from the pacemaker. ("Heart," 1909–10, i. 265. T. Lewis.)

In cases of simple paroxysmal tachycardia the electrocardiogram will show an abnormal auricular complex, because the stimulus originates outside the sino-auricular node. As, however, the stimulus reaches the ventricles by the normal channel, the ventricular complex will be normal. Fig. 48, which shows the end of a simple paroxysm of tachycardia, illustrates these points.

Clinical Diagnosis.—It occurs at any age after the first decade, but, according to Lewis's statistics, appears to be commonest between the ages of twenty and thirty.

The male sex suffers more frequently than the female.

These attacks may give rise to very little discomfort, or, if they last for only a few minutes, pass unnoticed, but if they last over half an hour they are more perilous. The clinical picture will therefore vary very much according to the duration of an attack, the rate of ventricular contraction, and the condition of the heart muscle.

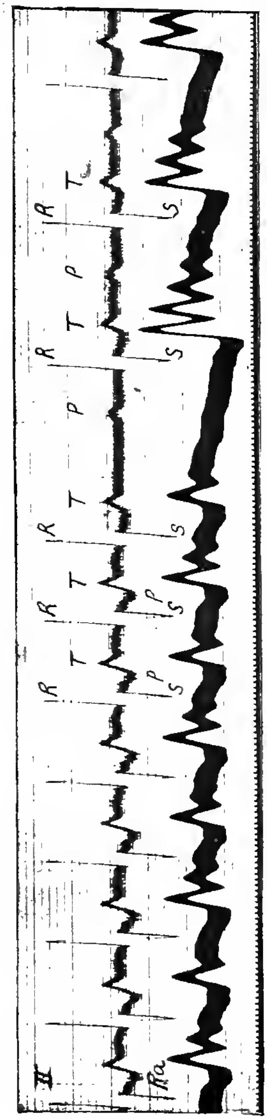


FIG. 48.—THE END OF A SIMPLE PAROXYSM OF TACHYCARDIA WHICH AROSE IN THE AURICLE.

There is a delay in conduction during both slow and fast periods; during the latter the auricle contraction falls with the preceding ventricular systole. During the paroxysm the pulse shows alteration. There is also a slight alternation in the heights of R summits, the larger R corresponding to the smaller pulse beat. (From "Clinical Electro-Cardiography." T. Lewis.)

The patient has a sense of discomfort in the chest or a slight fluttering, feels exhausted, cold, and sweaty. Then gastric symptoms may set in, with flatulence, salivation, nausea, and vomiting. Anginal attacks occur and vary much in severity, with precordial pain, hyperalgesia, and hyperæsthesia, tender muscles, and sense of constriction round the chest. The pulse is usually rapid up to 180 or even 200 per minute, and regular; ordinary changes of position do not affect the rate; the heart dilates, its sounds are tic-tac; murmurs if present disappear; there is large venous pulsation and congestion in the neck; the patient becomes first pale and then cyanosed, the liver enlarges, so also may the spleen. The condition frequently goes on to ascites and general anasarca, when death may follow, but usually the attack suddenly ceases, when the patient immediately feels extraordinary relief, a large amount of urine is passed, and all the symptoms and physical signs disappear amazingly quickly. The onset and offset of these attacks is sudden, and recognised by the patient, which is an important point in diagnosis.

This rhythm differs from auricular flutter in that the ventricle responds to each auricular stimulus, and that an attack has not been known to last longer than a fortnight.

4. AURICULAR FLUTTER.

This really must be looked upon as a condition of multiple auricular extra systoles, the new focus of stimulus production assuming rhythmical properties. The auricular rate is in excess of that of simple paroxysmal tachycardia, but the ventricular rate is usually less, because there is usually present a degree of heart block; the ventricle responding to only every alternate or even third auricular stimulus. The pulse is said to be usually regular, but it is frequently irregular, owing to the number of auricular contractions to which the ventricle responds, varying from time to time; but even then there is usually some system about the irregularity. This, however, is by no means always the case, as Figs. 49 and 50 show. When this grotesque form of irregularity is present, from an examination of the radial pulse only it would be mistaken for auricular fibrillation. Both Figs. 49 and 50 were taken from the same subject. An examination of the radial pulse in each tracing at once suggests auricular fibrillation. But a more careful examination of these two tracings will show that the condition is one of auricular flutter and not of fibrillation.

In both Figs. 49 and 50 point 3 usually but not invariably happens to fall at the beginning of a wave that might be thought to be carotid. Point 6, Fig. 50, sometimes falls at the summit of a wave that might be taken as a V wave, but it sometimes occurs also at the bottom of this wave. Then note that in Fig. 50 some of the largest waves in the tracing—marked *a*—occur during ventricular diastole in such a position, and in such numbers, that they cannot possibly be regarded as a single normal A wave occurring before a normal C wave. These waves can be explained only by regarding them all as auricular in origin. We are, therefore, driven to the conclusion that the case is one of auricular flutter, with a pulse irregularity of unusual type. The auricle is contracting at the rate of 300 per minute and the ventricle at 100 per minute.

In the electrocardiogram the auricular complexes are abnormal because the stimulus originates outside the sino-auricular node. All the auricular complexes in any one lead will have an exactly similar outline, but the outline of the P wave will vary in different leads. The ventricular complex is normal because the stimulus reaches the ventricles through the auriculo-ventricular bundle and its branches. Fig. 51 shows the three leads in a case of auricular flutter.

Clinical Diagnosis.—This condition has not yet been recognised long enough nor a sufficient number of cases recorded to determine definitely its age incidence; but it appears to be a condition of advancing years which affects the male sex more often than the female.

Many cases of flutter cannot be recognised with certainty without the aid of the polygraph or electrocardiograph. Usually the auricle beats between 260 and 320 per minute and the ventricle at half this rate or less—*i.e.*, there is a condition of 2 : 1 or even greater degree of heart

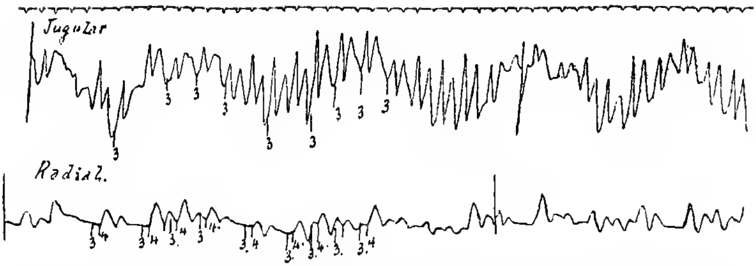


FIG. 49.—SIMULTANEOUS TRACINGS OF THE JUGULAR AND RADIAL PULSES, SHOWING AURICULAR FLUTTER, WITH A VERY IRREGULAR RADIAL PULSE.

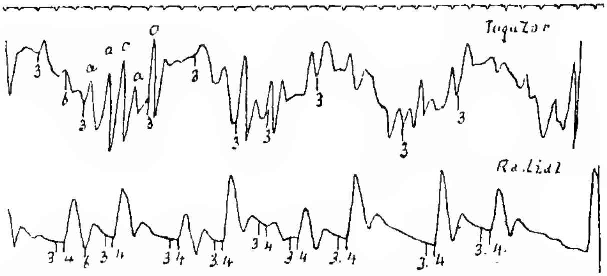


FIG. 50.—SIMULTANEOUS TRACINGS OF THE JUGULAR AND RADIAL PULSES, SHOWING AURICULAR FLUTTER, WITH A VERY IRREGULAR RADIAL PULSE.

From the same case as Fig. 49.

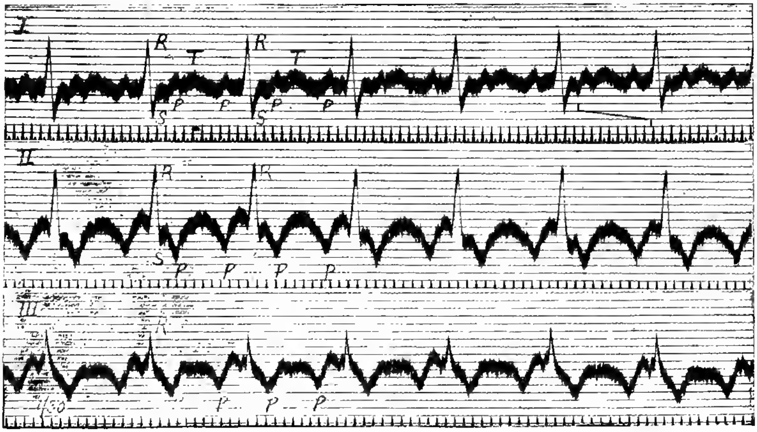


FIG. 51.—CURVES FROM THE THREE LEADS IN A CASE OF AURICULAR FLUTTER.

The auricular rate is 324, the ventricular rate is 162 per minute. In Leads II. and III. the auricular complexes are contiguous, and bear a constant relation to the beginning of R. From "Clinical Electro-Cardiography," T. Lewis.

block present. If the ventricle assumes the rate of the auricle the life of the patient is in great peril. The pulse is usually regular, but if irregular, as a result of changing from one degree of heart block to another, there is usually a distinctive pattern in the irregularity, and it does not usually assume that grotesque form seen in auricular fibrillation. However, the tracing already shown is in striking contrast to this statement (see Fig. 49).

The pulse-rate is usually 130 to 160 per minute. If the rhythm is not permanent the change in the pulse-rate is sudden during the onset and offset of the attack. The rate of auricular contraction does not vary with posture.

It is said that firm pressure on the carotid sheath, on the left or right side, sufficient to obliterate the vessel and to stimulate the vagus nerve, always produces a conspicuous slowing of the pulse or lapse of many beats caused by depression of conductivity of the *a–v* bundle. Short attacks of auricular flutter have the same clinical symptoms as simple paroxysmal tachycardia.

"Clinical Disorders of the Heart-Beat," by T. Lewis, 1915.

5. NODAL RHYTHM.

Nodal rhythm can be looked upon as a condition of multiple nodal extra systoles which are continued so as to constitute a rhythm. The stimulus giving rise to contraction of the heart is regarded as first making itself manifest in the junctional tissue probably in the *a–v* node. This may be due to either the stimulus material being more rapidly formed here than at the normal pace-maker of the heart, or to this tissue becoming more excitable than the *s–a* node.

Mackenzie first applied the term nodal rhythm to what we now know to be auricular fibrillation. He had noticed when the pulse assumed that characteristic

Quart. Journ of Med., 1907–8, i., 39-48.

irregularity which we now associate with auricular fibrillation that there was a disappearance of all evidence of contraction of the auricle, both clinically and in the jugular tracing, and he suggested that this was due to the auricle and ventricle contracting at the same time. In 1910 Lewis proved experimentally that the absence of all evidence of auricular contraction was accounted for by fibrillation of the auricles. True nodal rhythm is now confined to that condition in which both auricle and ventricle contract sumultaneously but prematurely.

Heart, 1910, vol. i., p. 306.

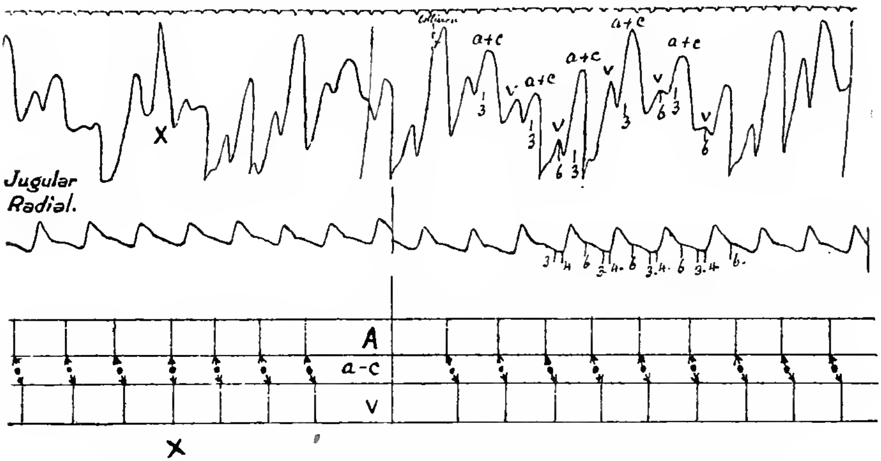


FIG. 52.—SIMULTANEOUS TRACINGS OF JUGULAR AND RADIAL PULSES AND KEY DIAGRAM IN A CASE OF NODAL RHYTHM.

The key diagram represents the stimulus as originating in the *a–v* node and spreading up to the auricle and down to the ventricle.

In Fig. 52, which is a tracing from a case of nodal rhythm, instead of the usual *a* and *c* waves in the jugular tracing there is one wave marked *a*+*c*. This wave, it will be noticed, is very large, and so bears strong evidence that the auricle and ventricle contract simultaneously. The key diagram shows a short space between the beginning of auricular and ventricular contraction, but in no case does this space exceed $\frac{1}{10}$ second, and this is too short a time for the contraction of the ventricule to be in response to a normal auricular stimulus. Moreover, there is precise proof that the ventricular contraction is not in response to a normal auricular stimulus in this tracing, for in the key diagram both auricle and ventricle begin to contract simultaneously at the place marked X. The corresponding wave in the jugular tracing, which is also marked X, is narrower than the other waves, in which the beginning of auricular and ventricular contraction is not quite simultaneous. The measurement of the interval between the beginning of auricular and ventricular contraction can be checked by Griffith's measurement from summit of A to beginning of radial rise. Choosing for accuracy those $a+c$ waves with a sharp summit, in no case does this measurement exceed $\frac{1}{10}$ second, and in the beat marked X it shows that auricle and ventricle begin to contract simultaneously. It will be observed that the rhythm is regular. In the key diagram the stimulus is represented as originating in the junctional tissue and travelling in both directions—*i.e.*, up to the auricle and down to the ventricle.

Before diagnosing nodal rhythm or nodal extra systoles from the electrocardiogram, the following four conditions must be satisfied:

1. Both the auricular and ventricular complex are premature.

2. If P is not lost in the Q R S deflections, but occurs as a separate wave, it is inverted. This is because the stimulus travels over the auricles in the reverse to its normal direction.

3. When P occurs as a separate wave, the P–R interval is less than the normal.

4. Since the stimulus reaches the ventricle by the normal

route—*i.e.*, by the junctional tissue—the ventricular complex is normal.

The last three beats in Fig. 53 are probably nodal in origin.

Clinical Diagnosis.—This condition cannot be diagnosed clinically with certainty; but the presence of large venous waves in the neck which are not periodic, but continuous, should raise a suspicion of nodal rhythm.

The first heart sound may be augmented.

If an auriculo-systolic murmur is known to have been present, but has disappeared, it should suggest nodal rhythm or auricular fibrillation. The pulse as a rule is regular in nodal rhythm and irregular in auricular fibrillation.

6. AURICULAR FIBRILLATION.

"The Study of the Pulse," 1902.

Although some of the earlier workers had noted cases with gross irregularity of the pulse, and others had observed marked systolic pulsation in the veins of the neck, Mackenzie, in 1902, called attention to the rule that a continuously irregular pulse is associated with

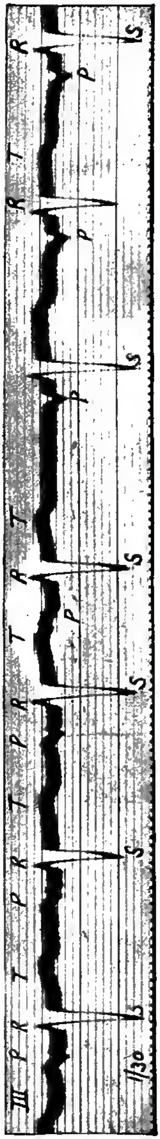


FIG. 53.—FROM A PATIENT WHO PRESENTED HYPERTROPHY OF THE LEFT VENTRICLE.

A regular rhythm of sinus origin is disturbed by a single premature beat arising in the auricle. Following upon the pause a rhythm of ectopic auricular origin is shown; there is some shortening of the P–R interval in these last three cycles; the new rhythm probably arose from the lowest level of the auricle—namely, the auriculo-ventricular node. (From "Clinical Electro-Cardiography." T. Lewis.)

the ventricular form of venous pulse. At first he believed that the ventricular form of venous pulse occurred because the auricle was paralysed from an overdistension caused by incompetency of the valves. In 1903 Hering gave a description of the character of this grotesque form of irregular pulse, and named it "pulsus irregularis perpetuus." A few years later Mackenzie abandoned his belief that the auricle was paralysed. In those cases in which gross irregularity of the pulse was associated with the ventricular form of venous pulse he held that the stimulus for contraction originated in the *a–v* node and passed both upwards and downwards, causing a simultaneous contraction of auricle and ventricle. He explained the absence of any manifestation of auricular activity in the venous tracing as the result of this simultaneous contraction of auricle and ventricle and gave to this condition the term "nodal rhythm."

Prag. med. Wochenschr., 1903, xxviii., 377-381.

Quart. Journ. Med., 1907–8, i., 39-48.

Lewis, working with the electrocardiograph, succeeded in demonstrating that in this clinical condition, which he named auricular fibrillation, the stimuli arise from a large number of different foci all over the auricular wall, so that the chamber as a whole does not contract but remains in the condition of diastole, while each fibre enters into fibrillary movements on its own account. The *a–v* bundle is unable to conduct such a great number of stimuli, so the ventricle responds to a fewer number, which pass along the *a–v* bundle at very irregular intervals. The number of stimuli to which the ventricle responds is usually greatly in excess of the normal rate, unless the patient be under the influence of digitalis.

Heart, 1910, vol. i., 306.

Figs. 54, 55, and 56, are typical tracings of auricular fibrillation. Each tracing shows a continuous irregularity of the radial pulse, and the ventricular form of venous pulse. As the auricle fails to contract, there are

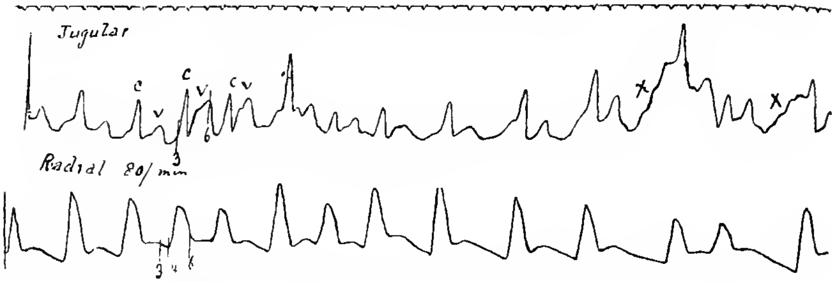


FIG. 54.—SIMULTANEOUS TRACINGS OF THE JUGULAR AND RADIAL PULSES FROM A CASE OF AURICULAR FIBRILLATION, SHOWING A CONTINUOUSLY IRREGULAR RADIAL PULSE AND THE VENTRICULAR FORM OF VENOUS PULSE, WITH A FEW FIBRILLATION WAVES MARKED X.

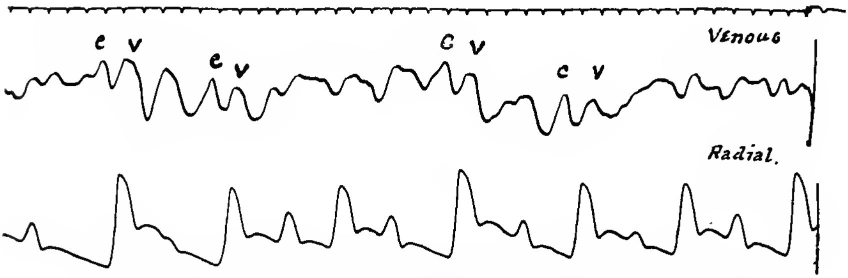


FIG. 55.—SIMULTANEOUS TRACINGS OF THE JUGULAR AND RADIAL PULSES FROM A CASE OF AURICULAR FIBRILLATION, SHOWING A CONTINUOUSLY IRREGULAR RADIAL PULSE AND THE VENTRICULAR FORM OF VENOUS PULSE.

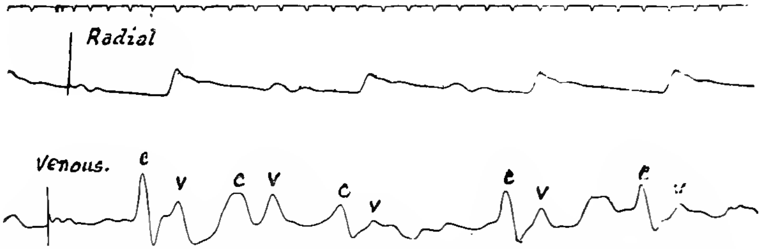


FIG. 56.—SIMULTANEOUS TRACINGS OF THE JUGULAR AND RADIAL PULSES FROM A CASE OF AURICULAR FIBRILLATION, SHOWING A CONTINUOUSLY IRREGULAR RADIAL PULSE AND THE VENTRICULAR FORM OF VENOUS PULSE.

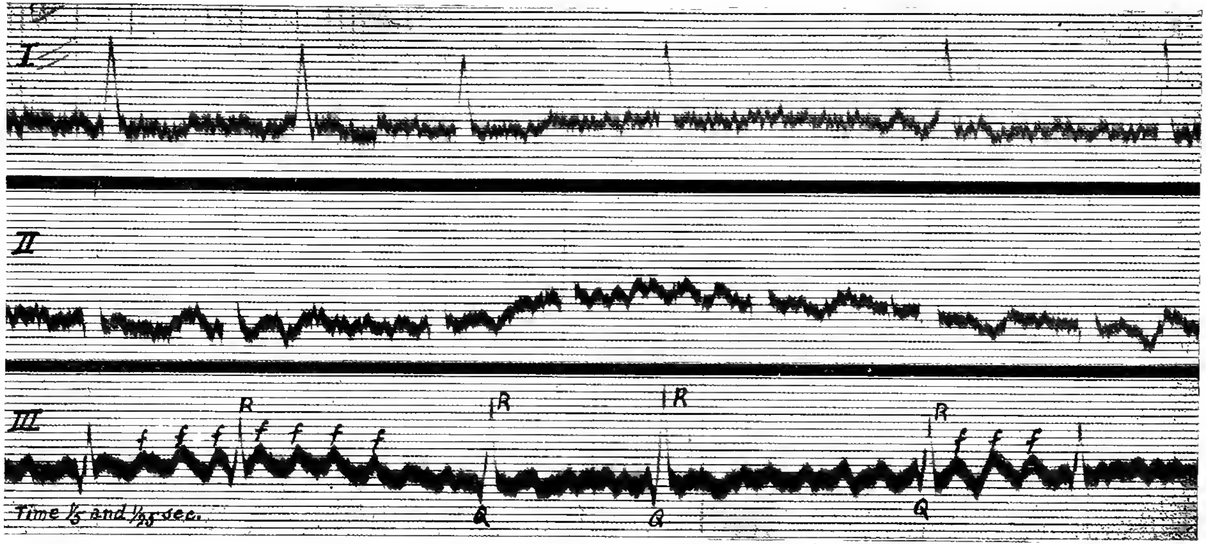


Fig. 57.—Curves from the Three Leads, showing Auricular Fibrillation.

The oscillations *f* are very well marked in Lead III. ; note they are not uniform and have not a constant relation to the beginning Q or R as in flutter. (Compare with Fig. 51.)

To face p. 146

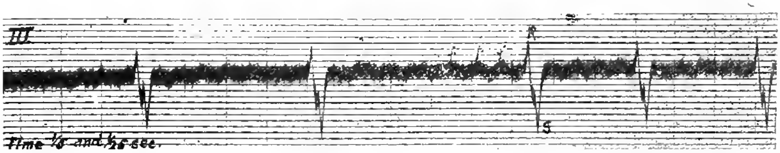


FIG. 58.—CURVE FROM LEAD III., SHOWING AURICULAR FIBRILLATION OCILLATIONS MARKED *F*.

Taken from the same case as Fig. 55.

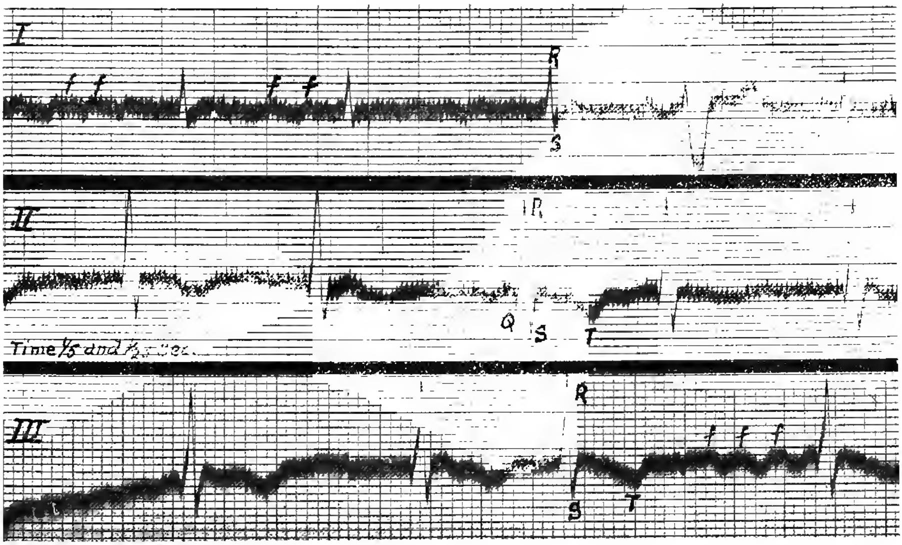


FIG. 59.—CURVES FROM THE THREE LEADS, SHOWING AURICULAR FIBRILLATION.

Taken from the same case as Fig. 56. (Incidentally Lead I. shows a right ventricular extra systole.)

To follow Fig. 57

no *a* waves in the venous tracing. Fig. 54 shows a few fibrillation waves marked X.

The electrocardiogram is the most certain means of diagnosing auricular fibrillation. There are no P waves, but during diastole there are a number of oscillations caused by irregular stimuli passing over the auricular wall. These oscillations have an average frequency of 500 per minute. They are never quite uniform, as are the P waves in auricular flutter; and, further, they do not bear a constant relation to the beginning of R. The stimuli which reach the ventricle travel by the *a–v* bundle and its branches, and therefore the ventricular complex is normal. Fig. 57 is an electrocardiogram, showing auricular fibrillation. The oscillations in lead III. are very large, but they are not uniform, and their relation to the beginning of R is not constant, as in flutter.

Fig. 58 was taken from the same patient as the polygraphic tracing, Fig. 55. Fig. 59 is also from a case of auricular fibrillation. It was taken from the same patient as Fig. 56.

"Clinical Disorders of the Heart-Beat," by Thos. Lewis.

Clinical Diagnosis.—For the purpose of studying age distribution, cases of fibrillation must be divided into non-rheumatic and rheumatic. Where there is no history of rheumatism the condition is distinctly one associated with advancing years, occurring most frequently in the sixth and seventh decades. In cases of rheumatic origin it is most frequent between the ages of twenty and thirty, and nearly as frequent between the ages of thirty and fifty.

The male sex is much more frequently affected than the female, but if the cases are divided into rheumatic and non-rheumatic, the sexes are found to be almost equally affected in the former series. This is on account of the frequent occurrence of mitral stenosis in rheumatic female subjects, this valve lesion being very commonly associated with auricular fibrillation.

It is estimated that 60 to 70 per cent. of all cases of heart failure with " dropsy " are suffering from auricular fibrillation.

The patient complains of a gentle but unpleasant sensation of fluttering in the chest, but rarely suffers from attacks of angina, except it be in some cases of mitral stenosis. The pulse is usually rapid, and exhibits a very grotesque form of irregularity, no two consecutive contractions being of the same strength, and the pause between the beats is continually changing, and bears no relation to the size of the contraction preceding it. This irregular pulse has been variously named as " the mitral pulse," " delirium cordis," or " pulsus irregularis perpetuus of Hering." The irregularity becomes more marked when the pulse-rate quickens, and this is in contrast to most other forms of irregularity, which become less irregular as the heart-rate increases.

Most irregular hearts, beating at a rate of 120 or more, exhibit fibrillation of the auricles. Sometimes when the heart is beating slowly it is impossible to detect the irregularity of the pulse in auricular fibrillation without the aid of a tracing and careful measurements.

As the weaker contractions of the ventricle do not force open the aortic valves, the irregularity is better observed by listening at the apex than by feeling the pulse. The character of the sounds is altered, as the second heart sound is absent after those ventricular contractions which fail to force open the aortic valves.

As mitral stenosis is very often associated with auricular fibrillation, the variability in these murmurs may be a help in the clinical diagnosis. Two factors must be borne in mind: that the auricle does not contract as a chamber, but remains in the position of diastole, and that the murmurs which occur during the diastolic period in mitral stenosis are dependent upon the rate of flow of blood from auricle to ventricle. When the auricle contracts, the inter-auricular pressure is greatest during the active contraction of this chamber, and again at the opening of the mitral valve. The two varieties of diastolic murmurs in mitral stenosis are due to these two causes. The position of the murmur caused by the contraction of the auricle depends upon the time relation of this contraction to the diastole of the ventricle. This murmur, which is crescendo in character and called auriculo-systolic, disappears in fibrillation, whereas the other diastolic murmur remains unaffected by fibrillation and bears a fixed relation to the second sound of the heart. The amount of the diastolic period which this latter murmur occupies will depend upon the rate of the heart and the degree of stenosis.

When once auricular fibrillation has set in it usually so persists that the irregularity is permanent and the pulse does not, as in other forms of irregularity, become regular during some of the day. If this rhythm is not permanent the change of pulse-rate during the onset and offset of the attack is sudden.

7. HEART BLOCK.

The first description of a case of heart block was probably Morgagni's account of the case of his fellow-citizen Anastasio Poggi, who suffered from what we now call the Stokes-Adams syndrome. Morgagni did not suggest that the auricles and ventricles contracted independently of one another; but that such a condition does occur was first described by Galabin in 1875. A

"De Sedibus et Causis Morborum," Alexander's trans., 1769, i., 192.

Guy's Hosp. Rep., 1875, 280-281. *Revue de Méd.*, 1885, xv., 161-173.

Zeitschr. f. klin. Med., 1900, xxxix., 293-304. *Deutsch. Arch. f. klin. Med.*, 1899, lxiv., 316-331.

Brit. Med. Journ., 1902, ii., 1411-1413; and *ibid.*, 1905, i., 519-522.

paper was published by Chauveau in 1885 in which he gave venous tracings to prove that the auricle and ventricle contracted at different rates and entirely independently of one another. Wenckebach and His were the first to suggest that the independent auricular and ventricular rhythm was caused by a lesion of the auriculo-ventricular bundle which blocked the stimulus in its passage from auricle to ventricle. In this country Mackenzie has been the pioneer in this as in other forms of abnormal cardiac rhythm. We have already seen, while considering the function of conductivity, that heart block had been produced experimentally by Gaskell, Engelmann, His, Erlanger, and others. As in the lower creatures, so in man, heart block is caused by depression of the function of conductivity of the *a–v* bundle from one cause or another. It may be merely a delay in the passage of the stimulus from auricle to ventricle, or the stimulus may

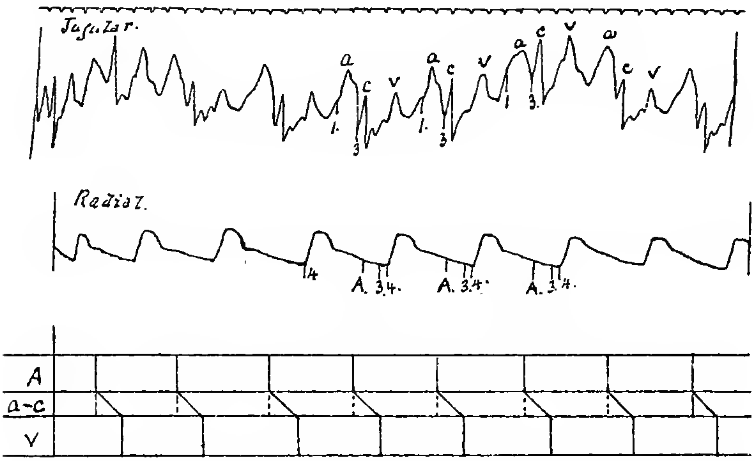


FIG. 60.—SIMULTANEOUS TRACINGS OF THE JUGULAR AND RADIAL PULSES WITH KEY DIAGRAM, SHOWING A LENGTHENING OF THE *a–c* INTERVAL, INDICATING PARTIAL HEART BLOCK.

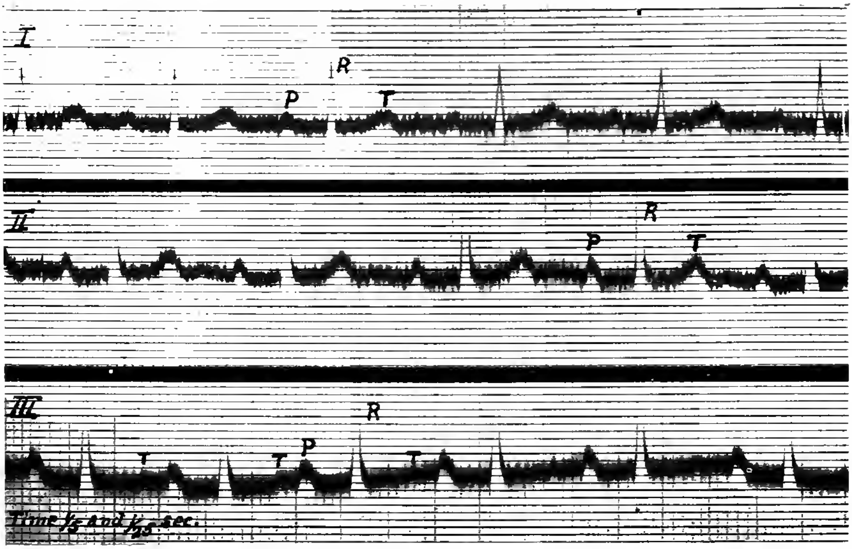


FIG. 61.—CURVES FROM THE THREE LEADS IN A CASE OF PARTIAL HEART-BLOCK, SHOWING A P.-R. INTERVAL OF OVER 1/5 SECOND.

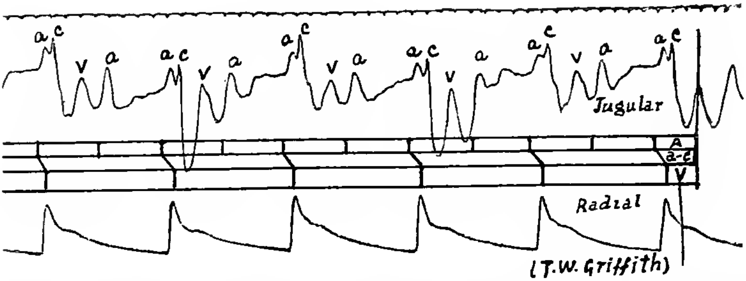


FIG 62.—SIMULTANEOUS TRACINGS OF THE JUGULAR AND RADIAL PULSES IN A CASE OF 2 : 1 HEART-BLOCK, SHOWING THAT EACH ALTERNATE AURICULAR STIMULUS FAILS TO FORCE A PASSAGE ALONG THE AURICULO-VENTRICULAR BUNDLE AND STIMULATE THE VENTRICLE TO CONTRACT.

To face p. 151

occasionally fail to pass from auricle to ventricle, in which case a ventricular contraction is missed; or there may be complete block, in which case none of the auricular stimuli passes to the ventricle, and this chamber contracts at its own slow rate to stimuli generated in the junctional tissue at some point below the auriculo-ventricular node.

Fig. 60 is an example of partial heart block in which every auricular stimulus gets through to the ventricle, but is delayed in its passage.

The *a–c* interval is a little more than one and a half fifths second. The interval is the same whether it is measured in the usual way or by Griffith's summit of *a* wave to beginning of radial rise. The summit of *a* wave is marked in on the radial tracing as A.

Compare this measurement of the *a–c* interval in this tracing with that in Fig. 17.

Fig. 61 is an electrocardiogram showing delayed conduction of the *a–v* bundle, the P–R interval being more than $\frac{1}{5}$ second. A further increase in the degree of block will result in some of the auricular stimuli failing to force a passage along the *a–v* bundle. This results in dropped ventricular beats.

Fig. 62 shows a tracing of 2 : 1 heart block, each alternate auricular contraction failing to stimulate the ventricle to contract. A still further development of the lesion results in complete heart block; in which both the auricle and ventricle contract independently of one another, each chamber contracting in response to its own stimulus.

Fig. 62, which shows 2 : 1 heart block, was taken by Dr. T. Wardrop Griffith on March 4, 1913. On October 27, 1918, I took Figs. 63 and 64 from the same subject. Both the polygraphic tracing and the electrocardiogram show that complete heart block is established.

Fig. 64 shows that the deflection P has no fixed relation to the ventricular complex.

Clinical Diagnosis of Heart Block.—This condition may occur at any age; the male sex is more commonly affected than the female. For the purpose of clinical recognition it is best to divide cases of heart block as follows, and to consider each separately:

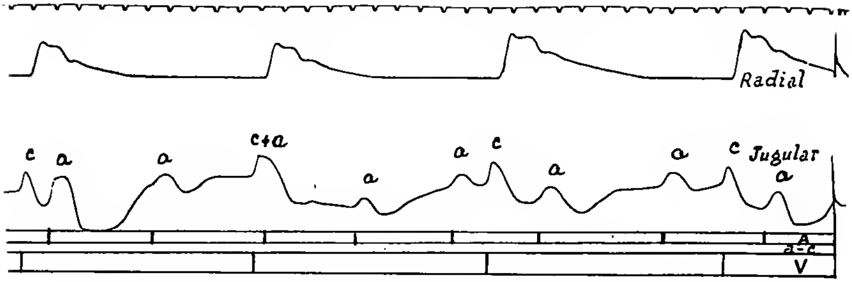


FIG. 63.—SIMULTANEOUS TRACINGS OF THE JUGULAR AND RADIAL PULSES, WITH KEY DIAGRAM, SHOWING COMPLETE HEART BLOCK.

The auricle and ventricle are contracting independently of one another. This tracing was taken from the same patient as Fig. 62, but five and a half years later.

(*a*) Lengthening of the auriculo-ventricular interval without any dropped beats.
(*b*) Dropped beats.
(*c*) Complete heart block

To consider these in the above order—

(*a*) This condition in some cases cannot be diagnosed except by the aid of a tracing from the polygraph or electrocardiograph; it may, however, give rise to two signs. It has now been established, by the aid of the microphone connected to a string galvanometer, that the contraction of the auricle gives rise to a sound. In the normal heart-beat it is not heard as a sound separate from the first cardiac sound; if, however, the space between the con-

"Lectures on the Heart," by Thos. Lewis.

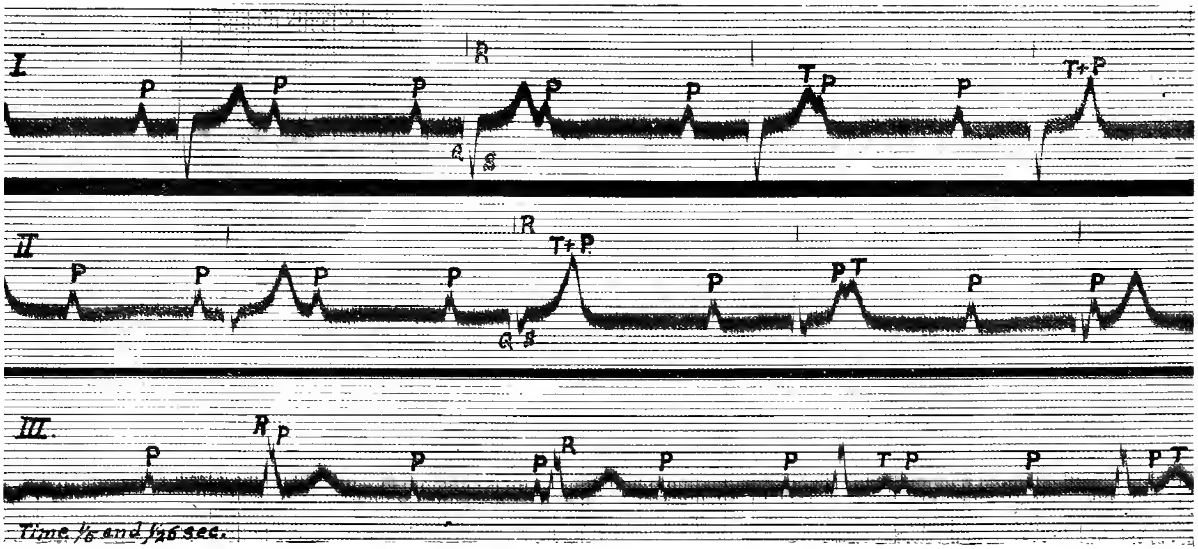


FIG. 64.—CURVES FROM THE THREE LEADS IN A CASE OF COMPLETE HEART-BLOCK, SHOWING THAT AURICLES AND VENTRICLES ARE CONTRACTING INDEPENDENTLY OF ONE ANOTHER.

The P. deflections have no constant relation to the ventricular complex. This electrocardiogram was taken on the same day and from the same subject as Fig. 63.

traction of the auricle and ventricle is increased, the auricular contraction may be heard and cause a reduplication of the first sound; or if the *a–v* interval is further increased, an apparent reduplication of the second sound.

In those cases of mitral stenosis in which the contraction of the auricle gives rise to a crescendo murmur running up to the first sound, if the *a–v* interval is increased, this murmur becomes separated from the first sound.

(*b*) Dropped beats. The single dropped beat in an otherwise regular pulse is not difficult to recognise. During the pause in the pulse there is no movement or sound at the apex, and the pause bears no constant relationship to respiration, as occurs in sinus irregularity.

When the dropped beats become more frequent and when definite rhythms are set up, such as a 2 : 1 heart block, it is impossible to diagnose this condition without the aid of graphic records. But 2 : 1 heart block should be suspected if the heart-rate is between 40 and 50 per minute.

During exercise the pulse-rate may suddenly increase to double its former rate and return again suddenly to that rate after the exercise is stopped. When the venous pulsation in the neck is well marked it will be found to be quicker than the ventricular rate.

In cases of mitral stenosis the murmur, due to auricular contraction, will stand alone in diastole, separated from the heart sounds, when the ventricle fails to contract; but it will precede the first sound when the ventricle responds to the auricular contraction.

(*c*) Complete heart block. This condition should be suspected when the ventricle beats regularly at 35 per minute or less. The rate is not affected by exercise. During diastole muffled sounds due to the contraction of the auricles may be heard at the apex which may give rise to reduplication of the first or second sound, or, when the auricles and ventricles contract together, to an intensified first sound. This intensification of the first sound, which was first described by T. Wardrop Griffith, forms a valuable bedside test.

Heart, 1911–12, iii., 143.

The venous pulsations in the neck can often be observed to be much more rapid than the ventricular contractions, and when the first sound is intensified there is an increased venous pulsation, as the auricle throws its contents into the veins instead of into the ventricle, as both chambers are in contractions at the same time. A rhythmical increase and decrease in the size of the venous pulse in the neck should immediately suggest complete heart block. In those cases of mitral stenosis with complete heart block in which the auricular and ventricular contractions have no fixed relation to one another, the murmur due to auricular systole is very variable. It may precede the first sound or stand alone in diastole, or be absent when the auricle and ventricle contract together.

8. THE PULSUS ALTERNANS.

This condition was first described by Traube in 1872. We shall have to distinguish between the true pulsus alternans and the pseudo-pulsus alternans.

Berl. Klin. Wochenschr., 1872, ix., 185-188 and 221-224.

The true pulsus alternans is due to failure of the function of contractility of the ventricular muscle resulting in an alternation in the size of the pulse-beats, while the distance from the beginning of the large beat to the beginning of the small is equal to or slightly greater than the distance from the beginning of the small beat to the beginning of the large. This spacing arrangement is said to be due to a delay in the appearance of the weaker contraction in the radial pulse.

Wenckebach, on the other hand, describes two varieties of true pulsus alternans:

"Arhythmia of the Heart," by Wenckebach (translated by Snowball).

(*a*) The distance from the beginning of the larger beat to the beginning of the smaller is slightly less than the distance from the beginning of the smaller beat to the beginning of the larger.

In this variety he affirms that contractility alone is at fault, and explains the spacing as because a small wave is transmitted through the artery with greater rapidity than a large one.

(*b*) The distance from the beginning of the larger beat to the beginning of the smaller is slightly greater than the distance from the beginning of the smaller beat to the beginning of the larger. In this variety, he asserts, both contractility and conductivity are at fault. The depression of conductivity is not confined to the *a–v* bundle, but occurs throughout the auricle, so that stimulus production at the *s–a* node must continue to be built up rather longer than usual before the auricle will respond. As the duration of the large systole is greater than that of the small, recovery of conductivity will be slower after the former than after the latter.

The variation in the size of the beats is readily accounted for by Starling's theory of contraction—*i.e.*, that the cubic capacity of the contraction depends upon the length of the muscle fibre. While contractility is failing a small contraction occurs which is too weak to empty the ventricle as completely as formerly; during the next diastole the ventricle not only receives the usual supply of blood from the veins, but it also contains the blood that the weak contraction was unable to expel; the muscle fibres are thus lengthened, and the next contraction will therefore be stronger. Since this strong contraction drives more blood into the aorta than the weak one, the aortic pressure is raised after the larger contraction, and the ventricle will contain less blood at the beginning of the small contraction than at the beginning of the large; the next systole, therefore, will be small because of the shortening of the muscle fibres, and the higher aortic pressure against which it contracts.

The outlook in this condition is bad, but, before a grave prognosis is given, it is necessary to bear in mind the following conditions, which may stimulate true pulsus alternans and must first be eliminated:

1. The pulsus bigeminus. This is a condition in which, if the premature contraction be strong enough to open the aortic valves, every alternate beat is an extra systole, or every third beat is an extra systole if the premature contraction be too feeble to force open the valves.

2. After an extra systole, or a weak ventricular contraction, the pulse may have an alternating character for two or three beats without our casting any reflection on the musculature of the ventricle, but if this alternation persists for more than two or three beats it must be regarded as a case of true pulsus alternans.

3. In certain cases of complete heart block in which every alternate ventricular systole coincides with an auricular contraction the ventricular output is reduced, both on account of the contracting auricle not allowing the ventricle to contract to its normal extent as it prevents the ventricle from pulling down the auriculo-ventricular junction to the normal extent, and because the ventricle does not contain as much blood as during a normal contraction, since, as it contracts at the same time as the auricle, it is deprived of that blood which is normally thrown into it by auricular systole. This diminution in ventricular output during every alternate contraction gives rise to an alternation of the radial pulse.

'Schorstein Lecture," 1912, by T. W. Griffith

4. A case in which an interpolated ventricular extra systole occurs after every second ventricular contraction, and is too feeble to reach the radial pulse, will give rise to an alternation of the pulse, because the contraction following the interpolated extra systole will be weaker than the contraction preceding the extra systole, on account of the short space of time which elapses between the extra systole and the contraction following it.

"Schorstein Lecture," 1912, by T. W. Griffith.

This condition can sometimes be recognised only by the increased *a–c* interval in the contraction following the interpolated extra systole.

5. The pulsus alternans occurring in abnormal rhythms, such as auricular flutter and fibrillation, is not to be regarded as necessarily of evil omen, but rather as a temporary exhaustion of the musculature of the heart due to its very rapid contractions.

Fig. 65 is a radial tracing showing the true pulsus alternans. Each period is of exactly the same duration.

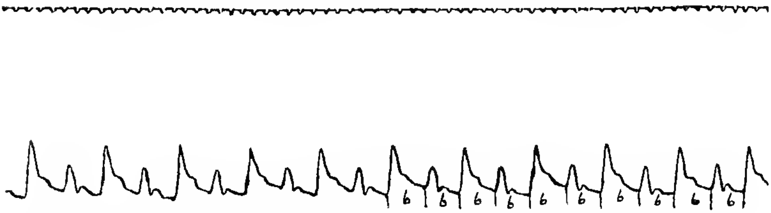


FIG. 65.—RADIAL TRACING SHOWING THE PULSUS ALTERNANS.

Each period is of exactly the same duration (six-tenths of a second). (From Mackenzie's "Diseases of the Heart." London: Oxford Medical Publications.)

This condition cannot usually be diagnosed clinically by feeling the radial artery. It can only be detected by the aid of a polygraphic tracing. The electrocardiograph does not reveal alternation of the pulse. Pulsus alternans should be suspected, and a tracing taken, in those patients who have regular pulse with a high blood-pressure and suffer from attacks of angina pectoris or cardiac dyspnœa.

9. SINO-AURICULAR BLOCK.

This condition is due to depression of stimulus production at the *s–a* node or delay in its explosive or flash point. It occurs in two forms: one in which there are intermissions of the whole heart, the other in which the rhythm of the whole heart is slowed. In this case the heart-rate suddenly may nearly double its rate in response to exercise, but after this any further increase is gradual.

Arhythmia of the Heart," by Wenckebach (translated by Snowball).

Wenckebach describes the block at the *s–a* node as due to depression of the functions of excitability or conductivity, or both, not in the *a–v* bundle but in the neighbourhood of the *s–a* node; hence a partial contraction, even if it takes place round this node, does not spread throughout the auricle and down to the ventricle.

This condition cannot be diagnosed without the aid of the polygraph or electrocardiograph. It is an uncommon condition and of little clinical importance.

10. ESCAPED VENTRICULAR BEATS.

In this condition the auricular stimulus is so delayed in its passage that the ventricle contracts in response to its own stimulus. The delay in the auricular stimulus may occur as the result of vagal stimulation depressing the *s–a* node, or due to depression of the conductivity of the *a–v* bundle, or it may be that the junctional tissue in the ventricle becomes more excitable than the normal pace-maker of the heart.

Since the stimulus to ventricular contraction arises in the junctional tissue, it is supraventricular, and therefore the ventricular complex of the electrocardiogram is normal. This condition cannot be diagnosed clinically.

THE SYSTOLIC MURMUR.

For many years there has been considerable difference of opinion as to the significance of the systolic murmur. After the outbreak of war the subject became one of very great importance to the army, because medical officers were recommending that soldiers should be discharged from the service solely on the evidence of a systolic murmur.

"The Soldier's Heart and the Effort Syndrome," by T. Lewis.

Lewis and his co-workers have carefully considered this subject and have come to the conclusion that, from the point of view of prognosis and treatment, no importance is to be attached to either apical or basal systolic murmurs, no matter what their character or direction of conduction. That is to say, on the one hand, if there is a systolic murmur and no physical sign of heart disease the murmur is ignored, and, on the other hand, if there is a systolic murmur and some physical sign of heart disease, such as cardiac enlargement, then the diagnosis of organic disease is based on the enlargement of the heart and the systolic murmur is ignored. We have worked upon this principle at the Heart Centre for the Northern Command with success. When, however, a definite purring systolic thrill accompanies a systolic murmur at either the apex or base, disease must be suspected even though no other sign of heart disease is present.

An uncomplicated valve lesion—*i.e.*, a diseased valve with a perfectly healthy heart muscle—would throw only a small additional burden upon the heart compared to the reserve force which a healthy heart muscle is capable of developing. The additional load due solely to the valve lesion is much less than is generally supposed. It has been shown that if an aortic valve is destroyed experimentally by an aseptic operation, the healthy heart muscle can cope with this additional load with little or no embarrassment.

The old school holds that valve disease produces heart failure by the additional load and internal stresses thrown on to the heart muscle.

The new school holds that valve disease is a certain proof of a diseased myocardium, and it is the infection or malnutrition of the heart muscle which causes the final breakdown.

The mitral presystolic and aortic diastolic murmurs, even when standing alone, signify respectively mitral stenosis and aortic regurgitation. The main significance of diagnosing these lesions is not that an estimate may be made of the additional burden they throw upon the heart, but rather as an indication that the heart muscle has been attacked by disease. It is not the valve lesion, but the damage done to the heart muscle which reduces the reserve force or produces heart failure. The systolic murmur standing alone cannot be used as an indication of disease of the myocardium in the same way as a mitral presystolic or aortic diastolic murmur can. We therefore ignore the systolic murmur from the point of view of prognosis and treatment. Lewis has illustrated by a good diagram the relation between the reserve force of a healthy heart muscle and the burden thrown upon it by a valve lesion. Fig. 66 is a reproduction of this diagram with his own description attached to it.

"The Soldier's Heart and the Effort Syndrome," by T. Lewis.

The accompanying diagram expresses, so far as I am able to express it diagrammatically, my conception of heart failure in relation to the load of work which the heart has to bear. I represent the capacity of the normal heart for work in the first column (A) of the diagram, dividing this column into two parts, to represent the load upon the heart while the body is at complete rest (*rest load*) and the *reserve*. The capacity for work in reserve I have intentionally drawn many times greater than the work of the heart muscle in conditions of rest. In the second column (B), I represent the heart fully loaded by exercise, putting forth the maximal quantity of blood against an increased resistance. I represent this *full exercise load* as many times greater than the *rest load*, but still show a reserve, though this is necessarily much diminished. In the third column (C) is represented a hypothetical instance of aortic reflux in which the muscle of the heart is supposed to be *perfectly healthy*, and I subdivide the whole column into the rest load, the aortic load, and the reserve. My objects in introducing this column are to represent the aortic load as no greater than the rest load and to show the reserve as little diminished. The reserve is still capable of absorbing the full exercise load without being itself completely exhausted (a condition represented in column D). Column D represents an ideal case, to which, however, certain instances of aortic reflux in soldiers closely approach, for the patients to whom I refer present an almost perfect exercise tolerance. Columns E and F represent these patients more truly, for in such aortic cases there is probably always some deficiency of the muscle; I represent the reserve as just exhausted by the full exercise load (column E), but as ample for all the usual calls of everyday life (column F). In column G, I represent the heart muscle as definitely deficient, the rest load and aortic load together occupying a considerable proportion of the whole column, and the reserve as greatly diminished as compared to the normal. Such a heart is incapable of meeting anything but small demands for increased work. Lastly, in column H, a heart is represented in which the muscle is very deficient and in which the rest load and aortic load are all that the heart is capable of carrying. Such a heart is just capable of maintaining the circulation when the body is at complete rest, but it is capable of no more.

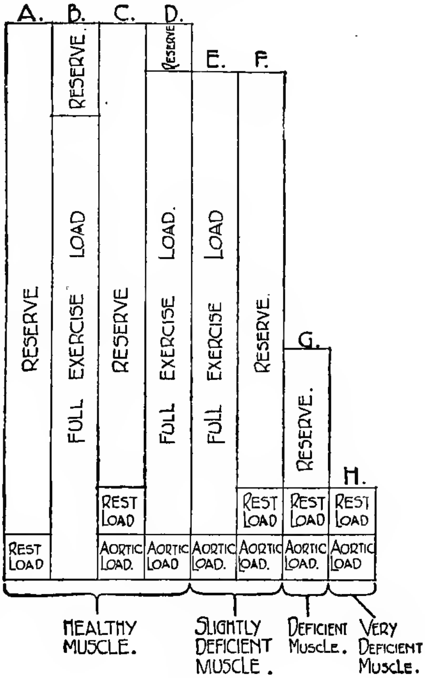


FIG. 66.

The object of the whole diagram is to stress the view that the heart fails, not so much because the load upon it is increased, but because its reserve is exhausted by damage of its muscle; that is my working hypothesis.

Before organic disease of the heart is diagnosed in a subject under forty years of age, one or more of the following twelve conditions must be present:

1. Mitral stenosis.
2. Aortic regurgitation.
3. Cardiac enlargement.
4. Abnormal rhythm.
5. Venous engorgement in the neck.
6. Aortic aneurysm.
7. True angina pectoris.
8. Pulsus alternans.
9. Chronic renal disease.
10. Constant high blood-pressure—*i.e.*, 160 mm. of Hg or more in a young subject.
11. A definite purring systolic thrill at the apex.
12. A definite purring systolic thrill at the base.

In a subject over forty years of age the myocardium cannot be pronounced as sound, even in the absence of any physical sign, if breathlessness and fatigue are present when at rest or on mild exertion.

THE PRINCIPLES OF TREATMENT.

I do not propose to enter into the question of drug treatment, but merely to point out how greatly the treatment of heart failure is affected by the discovery of the mechanism of the normal and abnormal cardiac rhythm, and that the method of treatment to be adopted must depend upon the character of the rhythm.

Whatever may be the various functions of the blood, that of the heart is to drive the blood into the various organs and tissues of the body and to keep them supplied with the necessary amount of blood required in the performance of their functions.

The force upon which the circulation depends is the musculature of the ventricles; failure of this muscle to supply the necessary blood to the organs and tissues is heart failure. Mackenzie has well described heart failure as "the condition in which the heart is unable to maintain an efficient circulation when called upon to meet the efforts necessary to the daily life of the individual."

"Diseases of the Heart," by Sir James Mackenzie.

The control of stimulus production at the *s–a* node in cases of "normal rhythm" is under the control of the central nervous system, and the increased heart-rate associated with heart failure must ordinarily be regarded as an attempt on the part of the heart to meet the demands of the tissues for more blood. The treatment of these cases must be to relieve the ventricle by lessening the demands of the tissues for blood, by making the patient rest or restrict his activities.

In cases of "abnormal rhythm," such as auricular flutter and fibrillation, the excitatory stimulus no longer lies in the *s–a* node and is no longer under the control of the central nervous system. The increased rate, then, in cases of abnormal rhythm does not serve any useful purpose, but, on the contrary, throws an extra burden upon the ventricle by driving it at an unnecessarily rapid rate.

Here the treatment is not simply to lessen the demands of the tissue for blood by resting the individual, but active steps must deliberately be taken to reduce the number of stimuli allowed to pass from the auricle to the ventricle. This can only be done by diminishing the conductivity of the *a–v* bundle through stimulation of the vagus nerves. This is effected clinically by the administration of the digitalis group of drugs; hence the wonderful efficiency of digitalis in the treatment of cases of heart failure with an abnormal rhythm.

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